Dental Management for Handicapped Persons in the Community

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Massachusetts Department of Public Health
Division of Dental Health

January 1985

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Supported by Division of Maternal and Child Health, Bureau of Health Care Delivery and Assistance, Department of Health and Human Services
MCJ 253329-01-0

In cooperation with the Massachusetts Health Research Institute, Inc.

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DENTAL MANAGEMENT FOR HANDICAPPED PERSONS IN THE COMMUNITY

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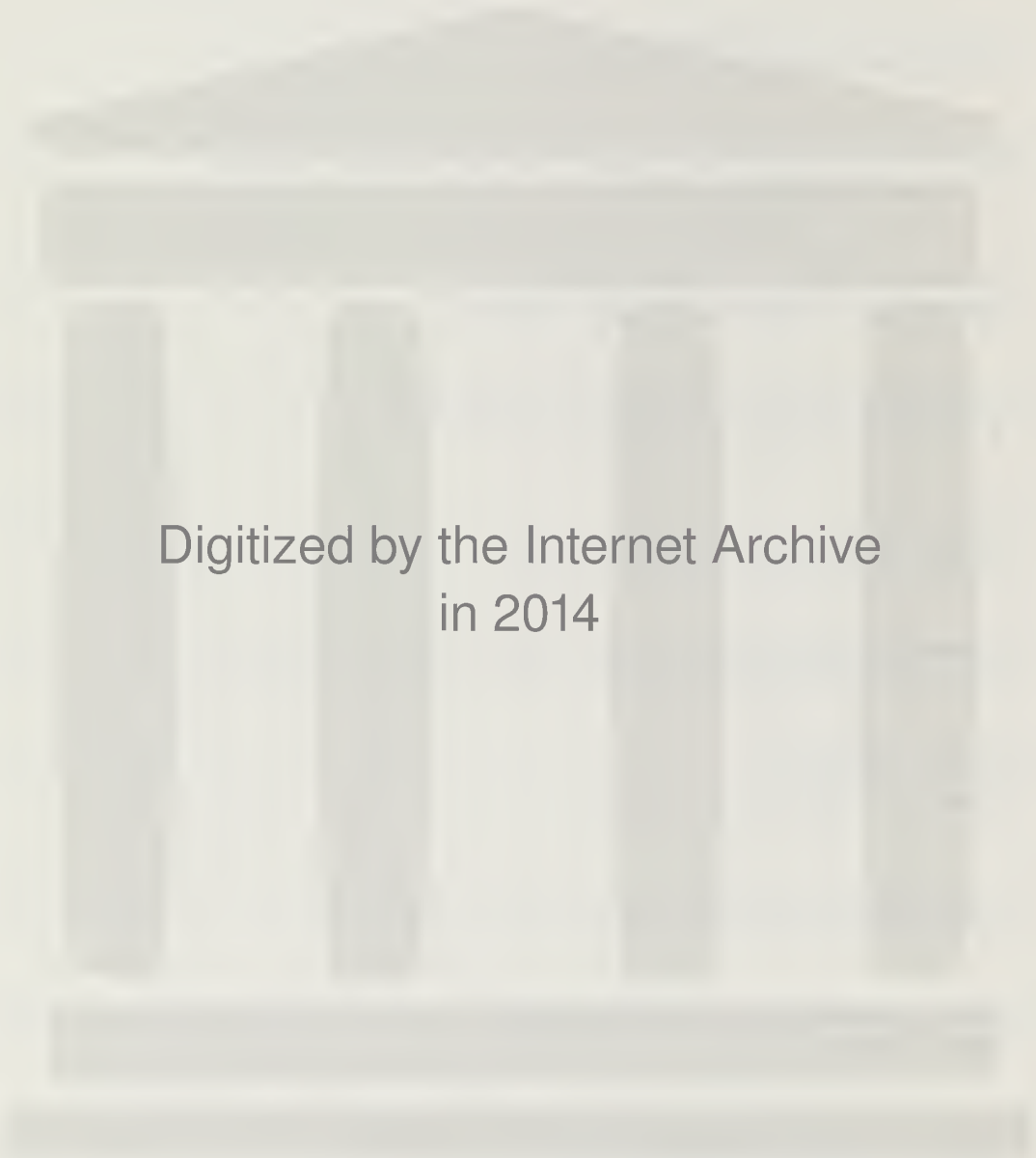

FORWARD

During the past ten years there has been a growing awareness of the need for developmentally disabled individuals to have greater access to dental care. Among the many barriers that have denied access to care has been the relatively small number of dentists who are trained in the special needs of this population. In the past, treatment of the developmentally disabled person was not an integral part of dental education. Dental schools have only recently recognized the importance of including didactic and clinical training in this area in the undergraduate dental curriculum.

This manual is designed by the Massachusetts Department of Public Health to offer the dentist basic information about current concepts in the dental management of the developmentally disabled person. No other group of patients challenge the knowledge and clinical skills of the dentist in so many areas, while simultaneously offering great rewards in the self-satisfaction of knowing that a critical need has been served. Hopefully, this manual will provide the catalyst for the reader to acquire further training and experience in treating this segment of our population.

Joseph P. O'Donnell, D.M.D., M.S.

SPECIAL ACKNOWLEDGEMENT

The Editor and production staff would like to acknowledge the exceptional contribution to this manual by the Department of Pediatric Dentistry at the University of Iowa College of Dentistry. In Chapter III, Dr. Arthur Nowak and his resident staff have compiled an extensive outline of the various types of developmental disabilities that a dentist may encounter in treating handicapped persons in the community. Each section defines the disability and provides information on the incidence, etiology, pathology, clinical and oral manifestations and diagnosis. Guidelines on treatment and dental considerations are also offered. The reader is thus provided with a thorough yet easy to read reference for greater understanding of the complex subject known as developmental disabilities.

Creation of this outline required many hours of literature review, research and clinical experience. Dr. Nowak and his staff are to be commended for their efforts in making this contribution to the dental literature.

DEVELOPMENTAL DISABILITY

Elizabeth Jones, B.A., M.S.

The purpose of this chapter is to share some facts about developmental disabilities and some thoughts about normalization. The "tone" of this chapter will be optimistic since there have been enormous strides in the field of developmental disability and there is much about which to be encouraged.

First of all, when discussing people with developmental disabilities it is important to recognize that when misunderstandings occur and a lack of knowledge prevails, the disability lies not in the individual with the impairment but in the community as a whole. The professionals experienced in working with people with developmental disabilities serve not only the consumer but will often be perceived as the "expert" or the teacher for neighbors and the community as a whole. Hence, these professionals become more important to public information efforts than any media campaign could ever be.

A developmental disability is defined by Public Law 95-602 as:

> "A severe, chronic disability of a person which is attributable to a mental or physical impairment or combination of mental and physical impairments; is manifested before the person attains the age of twenty-two; is likely to continue indefinitely; results in substantial functional limitations in three or more of the following areas of major life

activity: (self-care, receptive and expressive language, learning, mobility, self-direction, capacity for independent living, and economic self-sufficiency); and, reflects the person's need for a combination and sequence of special interdisciplinary, or generic care, treatment, or other services which are of lifelong or extended duration and are individually planned and coordinated."[1]

This statement is significant because it defines the disability in terms of a person's ability to function independently and in the context of a requirement for individualized, coordinated and extensive services. There is a dual focus on the person with the development disability and on others in the community.

Developmental disability also refers to mental retardation, autism, epilepsy, cerebral palsy and dyslexia when combined with one of the above. At the federal level and in some states, the addition of mental illness to the above conditions is being considered.

In terms of statistics, in Massachusetts, as of 1980, the general population is 5,778,840. The developmentally disabled number 87,838. Approximately 10,000 people have epilepsy and 22,948 have cerebral palsy. The Department of Mental Health

estimates that 10,157 have mental retardation with 3,800 in institutions and 6,357 living in the community. In the developmentally disabled population more than half are under the age of 18; 25% are from families below poverty level (only 11% of Americans fell into that category); 15% of those over age 15 have no formal education. Sixty-seven percent have income from public sources (while only 1% of American's income is from public sources); 62% have multiple disabilities.[2]

Clearly, there continue to be areas of economic and social dimensions which require critical growth in the next decade. Yet, it is important to keep in perspective the enormous strides that have been made through increased knowledge, technology, and most vitally, through advocacy by and on behalf of developmentally disabled children and adults since the early 1970's.

People with developmental disabilities have endured a number of role stereotypes that have severely limited their opportunities and abilities to achieve. Although three common role perceptions will be briefly mentioned below, it is important to remember that these characterizations exercised enormous control and literally determined the types of environments people with developmental disabilities lived and worked in; the training of the staff who were responsible for them; and ultimately the experience they were permitted or encouraged to undergo.

Firstly, there is a perception that these individuals are less than human. Certain terminology such as "vegetable" or "animals"

reinforce this perception. Direct care staff is viewed as "keepers" whose objective is containment in an environment that lacks esthetics and conventional comforts.

Secondly, there is a perception that these individuals are sick and diseased persons. Developmental disability is viewed as an illness with no cure, thus creating an aura of hopelessness. This stereotype fostered the early staffing patterns of institutions where staff was comprised primarily of nurses rather than specialized teachers and superintendents were physicians.

The third and most pervasive stereotype is that developmentally disabled individuals are eternal children. They are perceived as being incapable of accepting responsibility. This stereotype fosters a feeling of overprotection and a lowering of expectations for maximum development. This theory of normalization was first formulated in the United States in the early 1970's by Wolfensberger after progressive laws and enacted in Sweden and Denmark.[3] United States services had been primarily institutional and predominately of the warehouse variety with grass roots community services really just getting started by the efforts of parents themselves.

The definition of normalization is the use of means, practices and customs which are as highly valued as possible in order to promote and sustain positive and highly regarded behavior and characteristics. This basically presents a series of choices and actions. For example, the concept of integration versus

segregation must be considered along with age appropriate versus inappropriate behavior and responses. Although physical integration is the most focused upon aspect of normalization, social integration, i.e. people belonging to and participating in a community, is the goal.

There are a number of rationales of the importance of this concept. Role modeling is an excellent teaching technique and is more likely to work in an integrated setting. Also, integration allows access to valued peers rather than just other people with developmental disabilities. Furthermore, services and opportunities will be safeguarded when they are visible to all citizens and more individualized options are available for learning.

The task is to take these possibilities and use them. There is no magic per se in being in the community and one must not get trapped in the debate of institution versus community. Generic resources are a critical part of integrating developmentally disabled people into a community. It can open options for the individual with a developmental disability, enhance their status as citizens and educate the community about "differentness". However, it raises a number of serious issues that must be dealt with on a systemic level. First of all, there is the issue of accessibility with willingness of providers to serve people with developmental disabilities.

We must address the issue of incentives for providers who serve this population especially if there are high costs in time, equipment and knowledge. Are there enough providers with the knowledge to serve a person with a developmental disability in the appropriate manner? Is the service of acceptable quality?

Important challenges lie ahead and we are on the cutting edge of change. It is critical that we move forward to make ideals a reality in the services we provide to those citizens with developmental disabilities.

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PROFESSIONAL ATTITUDES TOWARD THE HANDICAPPED PATIENT

F. Edward Gallagher, D.M.D.

A generalized statement on prevalent attitudes in any field is often little more than an impression of personal experiences. It is subject to an immense number of variables not the least of which are the responses or feelings of others in the same field. The veracity of such a statement will always be subjected to conjecture, yet an examination of the dental profession's attitude toward the treatment of the developmentally disabled is important, for it draws out the good and the bad and, hopefully, clarifies the need of this group.

Historically, mental retardation was treated as a private subject. Its stigma was so great that, in the middle eighteen hundreds, Massachusetts opened a facility in Worcester to insure that these "out casts" would receive proper protection and care. This well meaning advent of "institutionalization" was to last one hundred years and coincided with society's shame. Parents could not acknowledge their children's difficulties and the health professions assisted with the proliferation of facilities where the handicapped could be cared for. It is unfortunate that what we now recognize as ignorance allowed the perpetuation of such a system. The very terms so descriptively a part of our language are indicative of this great misunderstanding. Bedlam and idiocy, lunacy and fits are all examples of a society that feared unexplained noises, unrecognizable behavior and the mysteries of a full moon. Witch hunts and burnings, more common abroad than here, were perhaps no more than a vilification of individuals with epilepsy. It is no wonder that society out of fear continued the incarceration of those who were different.

The 1960's initiated, slowly but steadily, the changes which we now accept. A president with first hand knowledge of retardation was instrumental in the passage of the Mental Retardation Facilities Construction Act of 1963. By 1967, nineteen university affiliated programs had been established to research and educate. The Developmental Disabilities Act of 1970 and our local chapter 766, signed in 1972, furthered the awareness. By then the terms "normalization" and "deinstitutionalization" were the buzz words of the field. The visibility of the handicapped increased dramatically. Parents kept and raised retarded youngsters at home while community residences brought previously "placed" adolescents and adults back to the mainstream where they sought normalized care. It is unfortunate, but perhaps historically understandable, that the system provided a less than enthusiastic response.

It is important to understand that the changes now affecting the lives of the disabled population were initiated by the same system that is often condemned for its callousness. An increasing awareness of the nature of mental retardation led to a discovery of potential and capacity that had, heretofore, been ignored. Certainly this realization began at the academic level, but its dissemination to the community at large was not far behind.

A philisophical schism has existed in our profession since the advent of these changes. Older, less informed attitudes of benign neglect have been confronted with an aggressive approach to treatment. Assuredly, those dealing in the area are aware of the dichotomy. It

would be simple and neat to consign the differences to age and education. Although these are contributing factors, we should look a bit deeper for a thorough explanation.

As a medical specialty, dentistry has progressed rapidly and forcefully in the last twenty-five years. We have produced larger numbers of graduates who cluster in the urban areas and who practice longer and more efficiently than our "cottage industry" predecessors. The technology of our profession alone has made it easier to treat all patients. From the high speed turbine handpiece to the visible light-cured composite resins, our "tools" have enabled us to render quality care in abbreviated periods. Many of the professional community have no understanding of the difficulties that existed in preparing a smile restoration with a belt driven handpiece and no local anesthetic. Coupled with the pharmacologic understanding of appropriate medication, the treatment of the handicapped patient has been greatly simplified. With government encouragement, we have also overcome many of the physical and financial barriers that had formerly prevented access to care. As a group, the dental profession is now providing substantial care of the retarded and handicapped population and yet we still have a long way to go.

Man is a complex animal. We can reason and we can rationalize. We can feel and we can fear. We are capable of working hard and hardly working. Somewhere from that maze comes the attitudes and the approaches we have towards our patients. As a profession we are and will continue to be faced with the challenge of providing suitable

health care in an empathetic way. The system demands this care, the parents seek this care and the handicapped deserve it.

Dentistry must look to four areas where attitudes allow barriers to care to be erected. Primarily, we must address education. Certainly, courses on the undergraduate and post graduate levels increase exposure, but didactics cannot replace "hands on" experience. It is important to understand the medical and sensory complications of dealing with handicapped individuals, but it is more important to have worked with them. Perhaps this is desensitization, but it would seem the most effective way of eliminating the fears indigenous to all unknowns. Dental schools are urban and have access to community-based programs. It would be wise to consider the efficacy of such an approach, for once the student has passed through the academic portrals, the opportunity is most probably lost.

Physical access to care is a second area of major concern. Elevators, ramps, wider doorways, and suitable equipment will eliminate both real and imagined barriers. They will diminish the excuses so easily rendered and they will enable our profession to provide the type of care that all groups deserve. The elimination of architectural limitations in conjunction with the profession's ability to use sedative and anaesthetic modalities in safe and appropriate settings will enable care to be delivered to all patients at all times.

The "idiological" barrier to dentistry for the handicapped may be the most difficult to overcome. The rationalization that quality care is a waste of time and effort must be discarded. Given proper education

and support, most handicapped individuals can sustain sound oral hygiene and good dental health. This is an area that much of dentistry has ignored for years, even with our most capable patients. Recently, the progress of the National Foundation of Dentistry for the Handicapped, among others, have demonstrated the fallacies of these misconceptions. These endeavors can prove that the time and effort of the dentist will be worthwhile and that the quality our profession is so proud of can be sustained. Educational programs for people at schools, community residences and training centers should be expanded to continue the progress already made. If we can demonstrate that dentistry for the handicapped can be as successful as dentistry in general, we can overcome a blockade that is "ideological" and a sustainer of benign attitudinal neglect.

The final and most visable area that impedes the delivery of care is financial. Many handicapped patients receive state support that reimburses a dentist at an "unacceptable" level. The system stigmatizes the patient twice. They are perceived as difficult to treat and unrewarding. This attitude is often quite wrong. Many of the handicapped patients are, frankly, quite easy to treat and provide rewards which, while not monetary, are more precious and more lasting. They are fun, they are different, and they are challenging patients. They alter the humdrum of our routine and they can give us a feeling of satisfaction that is hard to match. Certainly it would be appropriate to consider raising reimbursement levels for these individuals, yet to use this excuse to deny human service is equally as wrong. It is an

unconscionable restriction of care that cannot be justified and is uncompatable with a "profession".

Attitudes are very difficult to change, but it is not an impossible task. It is easier to mold new opinions from the outset and hopefully our profession will continue to succeed in this approach. However, having identified the components of some existing attitudes, we can perhaps work toward their modification. Dentists who avoid providing care for the handicapped are denying themselves and a most worthy group of patients. The areas that need attention are clearly defined. Ultimately, it is our responsibility to see that they are addressed.

A REVIEW OF TYPES OF DISABILITIES

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AUTISM

I. DEFINITION/DESCRIPTION

A. Autism - a severely incapacitating, life-long developmental disability characterized by disorders of language, socialization, and perceptual-motor difficulties.

B. The National Society for Autistic Children defines it as a syndrome with 4 essential features that are manifested before 30 months of age. Included are disturbances of:

1. developmental rates and sequences, involving delays, arrests, and/or regressions.
2. responses to sensory stimuli, including visual, auditory, tactile, vestibular, olfactory and gustatory, and proprioceptive disorders.
3. speech, language-cognition, and nonverbal communication.
4. capacity to relate appropriately to people, events, and objects, thus failing to respond appropriately to people and to assign proper symbolic meaning to objects.

C. Associated features vary with age, thought, mood, and behavior, such as labile moods, delusions, hallucinations, lack of appreciation for real dangers and self-injurious behaviors.

D. Autism occurs by itself or in association with organic diseases or metabolic disorders affecting the central nervous system (CNS).

E. Known as early infantile autism; aphasic, symbiotic infantile psychoses; childhood schizophrenia; Kanner's syndrome.

II. INCIDENCE

A. Rare syndrome identified in all parts of the world.

B. 4 to 5: 10,000 births.

C. More common in males, approximately 4 to 5 times more often.

D. Observed as early as 1 month of age but is most pronounced by 3 years of age.

E. In a small family, it is the first-born that is most often affected.

F. Some studies demonstrate an increased incidence with parents having a higher than normal intellectual status and socio-economic level.

G. Seasonal tendency, occurring more in late winter and early spring.

H. Frequently there is a history of perinatal complications.

I. No correlation between maternal age and increased incidence.

J. Unusual number of Down's syndrome relatives are found in autistic families.

K. Preconception histories show the parents of autistic children to have had a greater exposure to chemicals and higher incidence of hypothyroidism.

III. ETIOLOGY

A. Unknown at present.

B. Hypotheses that have been suggested:

1. Nuture theory-symptoms result from maternal deprivation. Parents are highly intelligent, emotionally cold people. Has not been shown to be true in recent studies.
2. Association with abnormal brain function, resulting in difficulty in processing afferent sensory stimuli in the dominant hemisphere.
3. Dysfunction of reticular formation of the brain stem, resulting in some pathophysiologic interaction of the visual and vestibular systems.
4. Possible biochemical problem, due to faulty metabolism of zinc, serontonin, indoleamine and tryptophan.
5. Damage to the CNS due to disease or injury, including phenylketonuria, maternal rubella, rhesus incompatibility, fetal anoxia,

prematurity, coeliac disease, lipodosis, lead poisoning, demyelinating disease, and chronic under-nourishment.

6. Possible autosomal recessive since approximately 2% of autistic children have an affected sibling.
7. A genetic and environmental combination, suggesting that an autistic child is the equilavent of a homozygote for high intelligence, associated with a special vulnerability to various complications during pregnancy and delivery.
8. An abnormal exaggeration of a normal defense mechanism against a reality which is either unrewarded or threatening to such a degree that the child loses motivation to become involved.

IV. PATHOLOGY

A. Most major pathologic findings are due to the child's inability to relate to people and things in the usual way from birth and obsessive desire for maintenance of sameness.

B. The repetitiveness and stability of behavior is suggestive of an ongoing pathological process within the CNS that is generating and maintaining the activity.

C. Pneumoencephalogram studies have shown enlargement of left temporal horn and ventricle.

V. CLINICAL MANIFESTATIONS

A. Developmental.

1. Delayed developmental milestones.
2. Retarded bone age.
3. Disordered cry.
4. Poor sucking and frequent regurgitation.

B. Cognitive, sensory, perceptual motor.

1. Mentally retarded.
2. Self-stimulating mannerism such as hand flopping, facial grimacing, and whirling.
3. Self-aggressive.
4. Poor coordination.
5. Hyperactive/hypoactive.
6. Spatially disoriented.
7. Problem with imitating.
8. Difficulty in perceiving objects just out of reach.
9. Unpredictable response to sounds.
10. Repetitive play.
11. Relative insensitivity to temperature and pain.
12. Increase in use of touch, taste and smell.
13. Mutism with normal hearing.
14. Nystagmus.
15. Reduced rapid eye movement (REM) activity.

C. Language:

1. Delayed onset of language.
2. Poor pronunciation.
3. Unusual vocalizations.
4. Echolalia.
5. Use of gestures to indicate needs.
6. Pronoun reversal.
7. Fragmented speech.

D. Problems with socialization.

1. Failure to assume the anticipatory posture when approached.
2. Resistance to being held.
3. Poor eye contact.
4. Inappropriate laughing and crying.
5. Frequent tantrums.

E. General appearance.

1. Physically attractive, healthy appearing child.
2. Facial expressions are intelligent looking.
3. An impression of being meditative and pensive is common.

IV. DIAGNOSIS

A. Symptons.

1. Extreme aloneness is the most striking symptom.
   a. child appears self-sufficient.
   b. parents have little meaning for them.

c. uninterested in conversation around them.

d. as infants, they seem apathetic.

e. withdraw from anything that disturbs their isolation.

f. relates well to objects.

g. sometimes treats parts of people as objects.

2. Language is always impaired.

a. 2/3 of children acquire some functional speech.

b. severe expressive language difficulty.

c. child appears deaf but hearing is normal.

d. words are used inflexibly and literally.

e. echolalia and delayed echolalia are common.

f. letters are reversed or omitted in speaking.

g. language problem due to inability to imitate or understand gestures.

h. improper use of personal pronoun.

i. some have marked preference for non-verbal language.

j. unable to use "yes"-indicates affirmation by repeating the question.

3. Obsessiveness is evident.
   a. there is an intense desire to maintain sameness.
   b. basis for ritualistic behavior.
4. Cognitive, sensory, and perceptual-motor problems.
   a. IQ varies.
      1. 60% have IQ below 50.
      2. 20% have IQ between 50-70.
      3. 20% have IQ above 70.
   b. Some possess "islets of ability".
      1. perform best on assessing manipulative or visual-spatial skills and rote memory.
      2. some have musical talent.
   c. An increase use of the senses of touch, taste, and smell.
      1. idiosyncratic eating habits.
      2. identify objects by licking or tapping them.
   d. Peripheral vision tends to be used in preference to central.

e. Avoidance of complex visual patterns-reason for avoiding adults.

f. Often insensitive to pain or temperature.

g. Unable to localize a sensation.

h. Great pleasure from swinging, rocking, sliding, riding and spinning.

i. Overactivity is common and becomes worse in complicated situations.

j. Seizure activity increases with age.

B. Tests.

1. Endogenous serotonin levels are often higher.

2. Serum zinc levels are usually elevated.

C. Differential Diagnosis.

1. Mental retardation.

2. Specific sensory deficits.

3. Congenital, developmental, and acquired disorders of central processing of language.

4. Sequelae of physical or psychological trauma.

5. Schizophrenia, childhood type.

6. Degenerative organic brain syndromes.

VII. TREATMENT

A. Multidisciplinary approach involving pediatrician, psychiatrist, neurologist, psychologist, teacher, social worker and family.

B. Treatment is primarily symptomatic.

1. Pharmacotherapy is of minimal benefit. The medications include a wide variety, such as megavitamins, neuroleptics, cerebral stimulants, sedatives, psychotrophic hormones, and lyseride (LSD).

2. Psychotherapy is currently the most useful technique available for helping autistic children.

a. Behavior modification-reinforcing or punishing a behavior in order to increase or decrease its rate of occurrence.

b. Relationship therapy-treating a child through a forced personal relationship.

3. Special education and speech classes to meet the child's individual needs.

C. Periodic medical, neurological, psychological, educational and behavioral reassessments.

D. Counseling therapy for the family.

VIII. PROGNOSIS

A. Poor for the majority but can be improved with a good educational program at school and home.

B. Much worse for children who are without useful speech at 5 years of age.

C. The worst for children with developmental receptive aphasia than for those with expressive defects only.

D. Poor outcome for those who have grossly abnormal EEG records.

E. 75% are classified as mentally retarded throughout life.

F. Few autistic children will recover.

G. Life span depends on whether there is a complicating organic disease. If there is none present, then a normal life expectancy can be anticipated.

H. In general, interpersonal relationships tend to improve as children grow older.

I. Aggression and self-injury tend to decrease with age.

J. If marked improvement is to occur, it is usually evident by age 6 or 7.

K. Improvement in learning and acquisition of skills frequently continues into adult life.

L. In general, most symptoms get milder as the child becomes older.

IX. DENTAL CONSIDERATIONS

A. Eating disturbances are present, such as idiocyncrasies and sameness in diet; holding food in mouth for extended periods of time; refusal of solid food, and preference for soft foods; preference for food stuffs high in sugar content.

B. Dental problems related to a high incidence of bruxism.

C. Increased rate of mouth trauma due to accident proneness and self-induced physical injuries.

D. Significant percentage have convulsive disorders, therefore, there is a problem with gingival overgrowth secondary to phenytoin.

E. Oral hygiene is poor because the child resists the assistance of another individual, and lacks appropriate intellect, communicative skills, and proper motor coordination.

F. Increased incidence of anemia, making them poor surgical candidates and demonstrating compromised gingival health.

G. Little self-cleaning action in the mouth due to poor tongue and cheek coordination.

H. Often a decreased salivary flow due to medication, thus increasing the chance for caries and periodontal disease.

I. Appropriate behavior is often reinforced with cariogenic foods at home and in school.

J. Oral hygiene is often a low priority because of the overwhelming attention these children demand from parents.

K. Many of these children are institutionalized for extended periods of time and receive minimal oral care.

L. Dental management of the autistic child is very challenging:

1. The dentist's contact with the child should be as noninvasive and nonthreatening as possible.
2. Gradual exposure to dental operatory is important.
3. Direct eye contact with verbal and nonverbal communication is essential.
4. Do not keep the child waiting long in the dental office.
5. Use only simple commands.
6. Ignore tantrums, self-stimulating and self-abusive behavior.
7. Movements should be slow, consistent and purposeful.
8. Small, repetitive steps may be necessary in order to approach the oral cavity.
9. Each dental procedure must include a simple demonstration followed by positive reinforcement.
10. Due to strong fear of the unknown, a family member should accompany child to dental office.
11. Parent or therapist working alongside the dentist may be helpful.

12. Rehearsal with dental props at home may be beneficial.
13. Minimal movements by the dentist and assistant should be made because the child is easily distracted and possesses a high degree of lateral vision.
14. Short time-outs are important so that tension does not build up, thus triggering an aggressive, fitful child.
15. Hand over mouth exercise (HOME) has only a very minimal benefit as an adversive stimulus due to poor communication skills.
16. An attempt should be made to lower noises, including conversation, oral vacuum systems, and high pitched handpieces.
17. Due to sharp swings in mood, reappointment of the child at the discretion of parent may be advantageous for both the patient and the operator.
18. Drugs for behavior control may be of some assistance. Hydroxyzine HCl, dyphenlydramine, diazepam, prothethazine and nitrous oxide analgesia are the agents of choice. Paradoxical reactions may occur with these mind altering drugs.

19. Treatment under general anesthesia in a hospital should be avoided because of the very marginal social adjustment of an autistic child. If hospitalization is necessary, the child should be exposed to only a limited number of personnel and a parent should be permitted to stay with child.

20. Many of these children have a disturbed pain threshold, therefore, frequent dental recalls are necessary to avoid a long standing dental infection.

X. PSYCHO-SOCIAL CONSIDERATIONS

A. Considerable emotional and financial stress of the family because child demands constant supervision.

B. Frequent sibling and spouse rivalry because they go to great lengths to monopolize their mother's time and attention by repetitive questions, self-injurious or provocative behavior, or tantrums.

C. Many of the children are healthy and attractive but oblivious to their emotional needs and those around them.

D. Socially, they can be very embarassing because they often mix normal with bizarre behavior.

E. Strong denial of the problem until signs become quite obvious that the child is not normal.

F. Delay in seeking expert help compounds an unhappy and confusing situation.

G. Parents are often guilt-ridden because they feel that they have emotionally deprived the child.

H. Tendency to reject the child if the situation becomes overwhelming.

I. Parents tend to become overprotective.

J. Parents often feel hostile toward professionals due to misdiagnosis or mishandling of the child.

K. Frustrations are compounded by inadequate community facilities for the child.

L. Families often become socially isolated due to the bizarre behavior of the autistic child.

XI. ECONOMIC CONSIDERATIONS

A. Financial burden due to the constant need for extended professional care.

B. Due to poor motivation, the autistic individual is not very productive in the work force.

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CARDIOVASCULAR DISEASES - SUBACUTE BACTERIAL ENDOCARDITIS (SBE)

I. DEFINITION/DESCRIPTION

A. Patients with congenital cardiac or vascular defects, rheumatic heart disease, and cardiac prostheses have a marked predisposition to developing a subacute bacterial endocarditis (SBE)

B. Bacteria identified may lodge in cardiac areas of scarring or stasis. Any bacteremia can potentially lead to SBE.

II. INCIDENCE

A. Infective endocarditis occurs in all age groups of infancy and childhood, including the neonatal period, with or without a recognized contributing factor.

B. Frequency of infective endocarditis appears not to have changed materially since introduction of antibiotics.

C. SBE is rare in children under the age of three.

III. ETIOLOGY

A. Congenital heart disease is principal underlying contributing factor.

B. Estimated to be present in about 75% of patients; those with Tetralogy of Fallot, aortic valvular anomalies, and ventricular septal defect appear to be especially vulnerable.

C. Cardiac surgery, recent or remote, is the most commonly identified precipitating event, especially when a structurally inadequate valve has been replaced with a prosthetic one or an aortic-pulmonary shunt has been created for palliation of congenital cyanotic heart disease.

D. Dental procedures, carious teeth, oropharyngeal surgery, instrumentation of the urinary tract or rectum and colon have been implicated as precipitating factors.

E. In neonates, as well as older infants and children, infective endocarditis may, on occasion involve a structurally normal heart, within or without an identified precipitating factor.

F. A wide variety of organisms have been identified as causative agents in infective endocarditis. Streptococcus viridans is most frequently identified, followed closely by staphylococcus aureus. Many other penicillin-resistant organisms may also be infecting agents.

IV. CLINICAL MANIFESTATIONS

A. Identification of infective endocarditis often based on a high index of suspicion in evaluation of an infection in a child with underlying contributing factor.

B. Symptoms include: History of predisposing heart disease. Usually insidious onset of chronic, low-grade

fever, anorexia, or malaise, but fever may be spiking. There may be a history of preceding infection (respiratory, skin, dental abcess, etc.) dental extraction, or tonsillectomy. Arthralgia, headache, chills etc. may also be present.

C. Onset may be acute and severe or mild and suggestive of a viral infection.

D. New or changing heart murmurs are common.

E. Serious neurologic complications may also be encountered.

F. Petechiae on skin, under nail-beds or in conjunctivae may occur.

G. Spleen may be palpably enlarged.

H. Myocardial abcesses may also occur with staphylococcal disease and may rupture into the pericardium.

V. DIAGNOSIS

A. Critical information for appropriate treatment of infective endocarditis is obtained from cultures or the blood.

B. Blood cultures must be obtained as promptly as possible in each child in whom infective endocarditis is considered a diagnostic possibility.

C. Blood culture should be repeated several times. If the clinical picture is suggestive of SBE, it is extremely important that the causative organism be identified.

D. Mild to moderate leukocytosis can be expected.

E. Erythrocyte sedimentation rate is commonly elevated.

F. A mild hemolytic anemia is not uncommon.

G. Microscopic hematuria may be present and is usually a manifestation of immune complex glomerulonephritis.

VI. TREATMENT

A. Ideally, treatment with one or more antibiotics selected on the basis of an in-vitro sensitivities of identified microorganisms to a variety of antibiotic agents.

B. Treatment can be initiated, after the infecting agent has been identified, without waiting for results of antibiotic sensitivity tests.

C. Bactericidal agents should be used whenever possible.

D. When the results of in-vitro sensitivity tests are available, appropriate changes in therapy are made if indicated and blood levels of the antibiotic are repeatedly monitored to insure that adequate anti-bactericidal levels are maintained.

E. In a desperately ill child or one who has recently had cardiac surgery or who is drug-addicted, antibiotic treatment should be initiated just as soon as initial blood samples have been obtained for culture, without awaiting the results. The antibiotics selected should insure coverage against penicillin-resistant staphylococci.

F. When no infective microorganisms can be isolated by culture, and a clinical diagnosis of endocarditis cannot be eliminated, treatment should be initiated. This should also be broad antibiotic coverage, including that for penicillin-resistant staphylococci.

G. In this case of penicillin allergy, vancomycin or a cephalosporin should be employed.

VII. PROGNOSIS

A. Despite availability of effective antibiotics and appropriate use of them, the case fatality of infective endocarditis in as high as 25% in some series.

B. Although eradication of infection is frequently accomplished, serious valvular disease may persist, and surgical repair may be necessary many years later.

C. When postoperative endocarditis develops as long as 3 to 6 months after cardiac surgery, disease is often curable with appropriate antibiotic therapy.

D. When complicating endocarditis is manifest in the immediate post-operative period, prognosis is grave.

G. Frequently, reoperation for removal of prosthetic material or replacement of artificial valves is required before the infection can be controlled.

VIII. DENTAL CONSIDERATIONS

A. Surgical procedures, endodontics, orthodontics, appliance therapy and even prophylaxis have produced a bacteremia.

B. Patients undergoing any dental treatment that may cause puncture or laceration of the gingival tissue or a pulpal manipulation that could produce a bacteremia should receive prophylactic antibiotic therapy. The American Heart Association recommends antibiotic prophylaxis with all dental procedures, including routine prophylaxis, that are likely to cause bleeding.

C. Teeth with a poor prognosis because of pulpal pathology or periodontal disease and are not responding to therapy should be considered for extraction because of their potential as foci of infection.

D. The prevention of SBE in patients with congenital heart disease is dentist's primary concern.

E. Oral infections should always be diagnosed and treated before heart surgery, if possible, so as to minimize the risk of postoperative infection.

F. The pediatrician or cardiologist should be consulted if a history reveals any unanswered questions.

G. Oral hygiene instructions and maintenance should be a major part in prevention since poor oral hygiene as well as periodontal infections may induce bacteremia.

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RECOMMENDATIONS FOR THE PREVENTION OF BACTERIAL ENDOCARDITIS

From the Council on Dental Therapeutics
Journal of the American Dental Association
January 1985

• Cardiac conditions.*

Endocarditis prophylaxis recommended
Prosthetic cardiac valves (including biosynthetic valves)
Most congenital cardiac malformations
Surgically constructed systemic-pulmonary shunts
Rheumatic and other acquired valvular dysfunctions
Idiopathic hypertrophic subaortic stenosis
Previous history of bacterial endocarditis
Mitral valve prolapse with insufficiency**
Endocarditis prophylaxis not recommended
Isolated secundum atrial septal defect
Secundum atrial septal defect repaired without a patch 6 or more months earlier.
Patent ductus arteriosus ligated and divided 6 or more months earlier
Postoperative coronary artery bypass graft surgery

*This table lists common conditions but is not meant to be all-inclusive.

**Definitive data to provide guidance in management of patients with mitral valve prolapse are particularly limited. It is clear that in general such patients are at low risk of development of endocarditis, but the risk-benefit ratio of prophylaxis in mitral valve prolapse is uncertain.

• Procedures for which endocarditis prophylaxis is indicated.

All dental procedures likely to induce gingival bleeding (not simple adjustment of orthodontic appliances or shedding of primary teeth)
Tonsillectomy or adenoidectomy (or both)
Surgical procdures or biopsy involving respiratory mucosa
Bronchoscopy, especially with a rigid bronchoscope*
Incision and drainage of infected tissue.

*The risk with flexible bronchoscopy is low, but the necessity for prophylaxis is not yet defined.

• Summary of recommended antibiotic regimens for dental/respiratory tract procedures.

Standard regimen	
For dental procedures that cause gingival bleeding, and oral/respiratory tract surgery	Penicillin V 2.0 gm orally 1 hour before, then 1.0 gm 6 hours later. For patients unable to take oral medications, 2 million units of aqueous penicillin G intravenously or intramuscularly 30-60 minutes before a procedure and 1 million units 6 hours later may be substituted
Special regimens	
Parenteral regimen for use when maximal protection desired (for example, for patients with prosthetic valves)	Ampicillin 1.0-2.0 gm intramuscularly or intravenously, plus gentamicin 1.5 mg/kg intramuscularly or intravenously, one-half hour before procedure, followed by 1.0 gm oral penicillin V 6 hours later. Alternatively, the parenteral regimen may be repeated once 8 hours later
Oral regimen for penicillin-allergic patients	Erythromycin 1.0 gm orally 1 hour before, then 500 mg 6 hours later.
Parenteral regimen for penicillin-allergic patients	Vancomycin 1.0 gm intravenously slowly over 1 hour, starting 1 hour before. No repeat dose is necessary

Note: Pediatric doses: Ampicillin 50 mg/kg per dose; erythromycin 20 mg/kg for first dose, then 10 mg/kg; gentamicin 2.0 mg/kg per dose; penicillin V full adult dose if greater than 60 lb (27 kg), one-half adult dose if less than 60 lb (27 kg); aqueous penicillin G 50,000 units/kg (25,000 units/kg for follow-up); vancomycin 20 mg/kg per dose. The intervals between doses are the same as for adults. Total doses should not exceed adult doses.

CEREBRAL PALSY

I. DEFINITION/DESCRIPTION

A. Characterized by paralysis, weakness, incoordination or any other aberration of motor function due to pathology of the motor control centers of the brain.

B. Aggregate of handicaps; emotional, neuromuscular, special sensory, and peripheral sensory, caused by damaged or absent brain structures.

C. Individuals who are handicapped by motor disorders which are due to non-progressive abnormalities of the brain.

D. A condition, not a disease.

II. INCIDENCE

A. Accurate reports regarding incidence are not available.

B. Incidence estimates range from 1.0 - 4.0 per 1000 live births.

C. 1 in 7 die within their first year of life.

D. Distribution of C.P.: 45.1% spastic; 23.6% athetoid; 12.5% rigidity; 10.8% ataxic; 3.4% mixed; 1.9% tremor; 1.7% rare cases.

E. 57% male; 43% female (marked similarity of distribution of types).

III. ETIOLOGY

A. Factors related to birth injury and postnatal developmental problems account for more than 50% of total cases.

B. C.P. following convulsions accounted for 9.3% of one population.

C. Interference with oxygenation of the brain (anoxia) is the common causative denominator. During the early prenatal period, the embryonic nervous system may suffer from oxygen lack due to a poor intrauterine climate as a result of neurotropic virus, infections, x-ray irradiation, or maternal illness of various types.

D. Clear-cut cases of cerebral palsy of genetic abnormality are rare. In one group of institutionalized cerebral palsy cases with severe mental deficiency, at least 10% were found to be genetically determined.

E. Other contributing factors - prenatal toxemia, Rh factor, infections, chronic maternal diseases.

F. In 656 reported cases, 134 mothers had experienced miscarriages or stillbirths either before or after the delivery of the cerebral palsied child.

G. It has generally been assumed that C.P. was related to the first-born child. This was assumed to be true because of the frequency of difficult labors in connection with first-born children. In a New Jersey study, only 24.5% were first-born. Second children accounted for 29.8%.

H. Perinatal Disorders: Interference with fetal and/or maternal respiration and circulation is a most important

cause of brain damage. Lack of oxygen supply to fetus arising from poor maternal blood supply - as would occur from anemia or hemorrhage, placental disturbance, or impairment of fetal circulation from twisted umbilical cord - is a common cause before the onset of active labor. Once the birth process is initiated, dynamic factors arising from uterine contractures, increased intracranial pressure from respiratory distress, and mechanical obstruction or trauma are responsible.

I. Neonatal and infancy periods, respiratory and circulatory difficulties most commonly interfere with oxygen supply, although infections and toxic and metabolic agents are frequently offenders.

IV. PATHOLOGY

A. No one characteristic pathologic finding.

B. Subdural hemorrhage due to traumatic dural-venous tears.

C. Spinal cord and brain stem damage due to mechanical injury.

D. Hypoxic damage to deep cerebral structures occurring predominantly in premature infant.

E. Hypoxic damage to cerebral cortex mainly in mature fetus and newborn infant.

F. Severe neonatal hypoxia may lead to total necrosis of cerebral structures resulting in porencephaly or hydrocephaly.

G. Moderate degree of anoxia produces less extensive destruction but varying degrees of severity of scarring and cystic damage to the cerebrum. This is often the basis for cerebral palsy or mental retardation.

H. A mild degree of anoxia results in focal or diffuse neuronal cerebral damage with consequent symptoms of "minimal brain dysfunction".

I. Hypoxic damage may be deep in area surrounding ventricles or may be more superficial in cortex.

J. Whether deep or superficial, damage to precentral gyrus produces spasticity; damage to frontal areas produces defects in mentality; damage to occipital lobe correlates with blindness or visual perceptual deficits; deep damage to the forebrain relates with athetosis and other forms of dyskinesia. With healing of damaged tissue, a scar may result causing seizures.

V. CLINICAL MANIFESTATIONS (General)

A. Relatively common to find physical and mental disabilities other than neuromuscular abberrations present in a given individual.

B. Clinical classification attempts accurately to describe type, location, degree and toxicity of primary neuromotor handicap and associated dysfunctions.

C. Clinical type based upon character of disordered movement.

1. Spasticity: 50-60%

   Motor function is impaired because of disharmony of muscle movements. Clinically - hyperactive deep reflexes, hypertonicity, abnormal plantar reflexes and clonus. The underlying component of spasticity is exaggerated contraction of muscles when subjected to elongation or "stretching".

2. Hypotonia (atonia): rare

   Muscles fail to respond to volitional stimulation. In C.P. some infants are hypotonic or "floppy" with absent deep reflexes during first year or so, therefore generally classified under spasticity category.

3. Dyskinesia (athetosis) 20-25%

   Involuntary extraneous motor activity accentuated by emotional stress.

4. Ataxia: 1-10%

   Incoordination due to a primary disturbance of balance, sense, posture, and/or kinesthetic feedback. Characterized by inability or awkwardness in maintaining balance with gross and/or fine motor incoordination.

5. Mixed: 15-40%

   Various combinations of the above types.

D. Topographic classification is used to complement clinical designation; various sites of neuromotor disability are:

1. Hemiplegia - findings lateralized to one half of body - 35-40%.
2. Diplegia - legs are more involved than arms - 10-20%
3. Quadriplegia - all four extremities are impaired - 15-20%.
4. Paraplegia - legs only - 10-20%.
5. Monoplegia - one limb is involved - rare.
6. Triplegia - three limbs are involved - rare.
7. Double Hemiplegia - both halves of body are involved - rare.

E. 35-60% C.P. have convulsions at some period during their natural life history.

VI. CLINICAL MANIFESTATIONS (Oral)

A. Conflicting opinions on greater or lesser incidence and prevalence of dental caries in the patient with cerebral palsy; most likely that the cerebral palsy population experiences caries at a normal rate.

B. Increased periodontal disease. Three factors are related to this increase:

1. use of diphenylhydantoin
2. bruxism - most common in athetoid form
3. poor oral hygiene

C. Occlusion. Abnormal muscle functioning is thought to be one of the major causes of malocclusion; abnormal muscle functioning such as facial grimacing, abnormal chewing and swallowing patterns and tongue thrusting; no difference in the distribution of occlusal types between the cerebral palsied and the normal populations studied; frequent minor tooth irregularities including open bites associated with abnormal tongue movements; protruding maxillary arches and incompletely covering upper lips are often seen in severely involved spastics; splayed maxillary teeth, accompanied by narrow constricted maxillary teeth, wide and relatively low maxillary arches are more frequently associated with the athetoid type.

D. Increased number of fractured anterior teeth due to trauma resulting from falls, mouthbreathing and enamel hypoplasia.

E. Hypoplasia - Teeth of cerebral palsied children exhibited altered crown morphology; can range from a chalky appearing area with an irregular surface through deep pinpoint defects in the enamel to crowns showing considerable loss of their enamel covering; 70% of the cerebral palsied cases having a defect showed a correlation between the location of the defect on the tooth and the time the insult occurred. The location of the defect on the tooth is useful in determining the time of insult to the fetus or child.

VII. DIAGNOSIS

A. Cerebral palsy can simulate a number of conditions so that often differential diagnosis is a difficult problem.

B. Babies with nutritional, metabolic, allergic, neurologic, orthopedic, psychiatric, and other related disorders are often referred to cerebral palsy clinics because of their irritability or developmental lag.

C. The younger child, the more vague the findings and prediction of outcome.

D. Newborn period:

1. Evidence at birth of anoxia does not guarantee later brain damage, nevertheless, the hyper-irritable or excessively listless infant must be regarded suspiciously for future signs of neurological dysfunction.
2. Hyper-irritability usually reflects cortical instability. The listless infant who is hypotonic, reflects mid-brain or brainstem damage.
3. A clue to continuing effects of anoxia is the persistance after birth of head lag.
4. C.P. is infrequently diagnosed at birth because there are few meaningful abnormal neurological signs at this time.

5. The brain-damaged neonate may appear normal for several weeks.

6. The visible signs during the early weeks of life suggesting future neuromotor disturbance may be cyanosis, pallor, stiffening, arching, excessive startle, strabismus, nystagmus, bruises, cephalhematoma and jaundice. Sudden unexplained fevers are not unusual.

7. Developmental stigmata such as uncleft toes, ear deformities, high-arch palate, hypertelorism, microcephaly or hydrocephaly, indicated early anoxic insults to the embryo or genetic effects may suggest the possibility of associated brain damage.

E. Infancy:

1. In a study of infants suspected of being C.P. -

   a. convulsions - 81%

   b. slow development - 54%

   c. feeding difficulties - 21%

2. Physical findings involve delayed motor development and variances from normal skeletal muscle tonus.

3. Classic reflex changes are unreliable indicators.

4. Tonus may be evaluated by palpitation of muscles to detect consistency.

5. Persistence of "brain-stem" derived activities beyond six months indicates a damaged or dysfunctioning cortex or lower main centers:
    a. occular signs (ciliary and pupillary reflexes)
    b. cardinal point signs
    c. Moro response
    d. grasp reflex
    e. other postural reflexes
6. During early months, deep reflexes may increase, increasing periods of hypertonicity, especially when limbs are moved.
7. Severely damaged infants, especially if mentally retarded, may show "mass movement", in which all of the extremities flex or extend simultaneously.
8. Spasticity generally appears before six months, whereas athetosis may not be apparent until the end of the first year or later. Spasticity affects the lower extremities more severely, while the head, neck and upper extremities are often more severely involved in athetosis.
9. The rate of developmental progress is useful in diagnosis. C.P. generally delayed in many areas (sitting without support, reaching for objects, crawling, etc...)

VIII. DIAGNOSIS (Laboratory Studies)

A. Lab tests are often not helpful although they are needed to be certain that correctable conditions are not present.

B. Transillumination of the skull in infants can aid immeasurably in detecting early surgically correctable abnormalities, as well as microcephaly with macrocranium, ventricular dilatation and porencephaly.

IX. TREATMENT

A. Management of C.P. demands comprehensive systematic approach.

B. The goal is normalization.

C. Orthopedic therapy to improve both functional mobility and appearance. Physical therapy, bracing.

D. Limited amount of orthopedic surgery.

E. Neurosurgery has not proved reliable but has been used in controlling dyskinesia, seizures and hemiplegia. (Removal of one cerebral hemisphere).

F. Pharmacological management of seizures (i.e. dilantin, phenobarbital).

G. Muscle relaxants to relieve muscle hypertonia. Especially valuable if it also favorably alters the child's emotional attitude and behavior. (Placebos often effective).

H. Behavorial medications: Medications that have a favorable effect on hyperkinetic behavior (Dexedrine, Benzedrine, Ritalin).

X. PROGNOSIS

A. Whether or not a child will make an adequate adjustment in later life can be answered by correlating:

1. History - absence of chromosomal or biochemical abnormality; absence of early severe prenatal disturbance.
2. Physical examination - absence of severe spastic quadriplegia.
3. Laboratory - absence of severe anatomic defect.
4. Psychological testing.
5. Psychosocial.

B. The pneumoencephalogram can be used as a rough prognostic indicator in cerebral palsy. The prognosis is poor in those cases with cerebellar atrophy and severe generalized cerebral or cortical atrophy; clinical progress is good in those cases with unilateral cerebral atrophy and when the pneumogram was essentially normal. The pneumoencephalogram of course does not take into account the multifaceted problem of personality, family, or social factors which contribute to ultimate success.

XI. DENTAL CONSIDERATIONS

A. Use of physical restraint and/or sedative drugs where indicated.

B. Cradle head in arm.

C. Use pillows, pads to accommodate limbs bent to awkward positions.

D. Consider G.A. if indicated.

E. The most important procedures are preventive in nature.

F. Positioning during brushing and flossing.

G. Dietary counselling.

H. Sealants, though efficacy has not been established, may be of use in coping with an extremely deleterious habit found occassionally in the cerebral palsied and other handicapped populations - rumination. Rumination is the ability to bring up stomach contents into the oral cavity at will. This leads to the dissolution of the enamel covering the teeth. SSC's may also help.

I. Sealant and composites for hypoplastic teeth.

J. Mouthguards reduce the wear from bruxism. Also to protect the teeth of children in crutch or brace training who are at risk of falling.

K. Many of these individuals may be mouth breathers which may contribute to poor gingival health.

XII. PSYCHO-SOCIAL/ECONOMIC CONSIDERATIONS

A. As in many other handicapping conditions, the family of a patient with C.P. may be under considerable emotional and financial stress.

B. Dentist must be supportive and understanding, and yet let the patient be as independent as possible (chair transfers, etc.).

C. Potential sources of financial aid should be sought out of local, county, state and federal levels.

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CYSTIC FIBROSIS

I. DEFINITION/DESCRIPTION

A. Hereditary disease of children, adolescents and young adults.

B. Due to generalized dysfunction of exocrine glands.

C. Chronic pulmonary disease, pancreatic deficiency, high sweat electrolyte levels, cirrhosis of the liver.

II. INCIDENCE

A. 0.5-1.0 per 1000 (2-5% of general population maybe carriers).

B. Rarely in Blacks or Orientals.

III. ETIOLOGY

A. Basic defect is unknown.

B. Deficiency in autonomic nervous system function due to absence of a necessary enzyme or other metabolic error has been proposed in general terms to explain the widespread dysfunction of exocrine glands.

C. Genetically transmitted (autosomal recessive).

D. A female with C.F. has a 1:46 chance of transmitting it to her child.

IV. PATHOLOGY

A. Mucous producing glands throughout the body may show accumulation of abnormal secretions leading to dilatation of the secretory organ.

B. Pancreas

1. Smaller, thinner and firmer than normal.

2. Obstruction of pancreatic ducts by abnormal secretions.

3. Dilatation of the secretory acini and ducts.

4. Degeneration of the exocrine parenchymia.

5. Pancreatic lesions are progressive.

C. Sublingual and submaxillary glands may be involved.

D. Gallbladder is small in 1 in 5 cases.

E. Localized foci of biliary obstruction in liver.

F. Obstruction of bronchi by abnormal secretions.

1. Leads to suppurative bronchitis.

2. Peribronchial pneumonia and diffuse obstructive emphysemia are common.

B. Nonmucous-producing glands (sweat glands and parotid glands) show no pathologic or histologic changes, although the chemical composition of their secretions is abnormal.

V. CLINICAL MANIFESTATIONS

A. Meconium ileus.

1. In 5-10% of patients with cystic fibrosis, intestinal obstruction occurs with consequent symptoms within hours after birth.

a. Perforation may occur.

B. Chronic pulmonary disease.

1. Present in almost all patients. (90% of patients)
2. Dry, nonproductive cough followed by secondary infection.

C. Clubbing of fingers and toes is common in chronically ill patients.

D. Chest is barrel shaped.

E. Radiographic evidence of generalized obstructive emphysema and bilateral parenchymal infiltration.

F. Involvement of paranasal sinuses - nasal voice, post-nasal drip.

G. Pancreatic insufficiency.

1. 80% of patients.
2. Gives rise to symptoms of intestinal mal-absorption
3. Malnutrition.
4. Abdomen is distended.
5. Stools are increased in number, bulky, greasy and extremely foul.

H. Cirrhosis of the liver.

I. Cystic fibrosis is the commonest cause of prolapse of the rectum in infancy and childhood.

J. Sudden rises in environmental temperature may result in massive salt loss through the sweat.

1. May lead to hyperpyrexia, coma and death.

K. Oral findings:

1. Cystic fibrosis is treated with antibiotics. If tetracyclines are used may cause discoloration for some or all of the primary and permanent teeth.

VI. DIAGNOSIS

A. Combination of symptoms of chronic pulmonary disease and of intestinal insufficiency suggests diagnosis.

B. Diagnosis should be based on the following criteria:

1. Increase in electrolyte concentration of sweat.
2. Absence of pancreatic enzymes.
3. Chronic pulmonary involvement.
4. Family history of disorder.

C. Sweat chloride test.

1. Up to the age of 20 yrs. a level of more than 60 mEq. per liter of sweat chloride is diagnostic of cystic fibrosis.

D. Pancreatic deficiency.

1. Examine duodenal contents for pancreatic enzyme activity if diagnosis is uncertain.
2. Tryptic activity is absent in over 80% of the patients.

E. Fecal examination - increase in free fatty acids (up to 30 g/day.)

VII. TREATMENT

A. The basic defect in cystic fibrosis is not known, and cure is not possible at present.

B. Treatment has four objectives:

1. General care of the patient.
2. Control of the pulmonary infection.
3. Maintainance of good nutrition.
4. Prevention or restoration of abnormal salt losses.

C. General care.

1. Continued immunizations against pertussis.
2. Multidisciplinary approach.

D. Control of pulmonary infection.

1. Use of antimicrobial agents.
   a. Selection of drug should be based on bacterial sensitivity tests.
   b. Penicillin and streptomycin generally have been the most effective.
2. Physical therapy measures to promote bronchial drainage.
3. Use of nebulizers with aerosol solutions to increase the hydration of secretions in order to facilitate expectoration.

E. Dietary therapy.

1. Should be high caloric, high protein and moderate in fat content.

2. Administration of pancreatic extracts.

F. Treatment of abnormal loss of salt.

1. 2-4 gm sodium chloride daily should be taken orally in hot weather.

2. IV saline solution in emergencies.

G. In meconium ileus, surgical intervention is imperative.

VIII. PROGNOSIS

A. Pulmonary involvement usually dominates the clinical picture and determines the fate of the patient.

B. Uncontrollable bleeding due to portal hypertension and massive loss of salt in hot weather are additional hazards.

C. Over 50% of affected children die before the age of 10.

D. Over 80% die before the age of 20.

XI. DENTAL CONSIDERATIONS

A. Poor anesthesia risk. Atelectasis, spread of lung infection. Avoid respiratory depressants, i.e. narcotics, major tranquiliers, even valium in severe patients. You want to prevent pooling of secretions, poor insulation.

B. Call physician re: physical status, even with N_2O-O_2. Chronic emphysema rely on low O_2 (O_2 lack) to stimulate breathing. Increase O_2 - stop breathing.

C. Breathing exercises during 3-hour appointment.

D. Expect coughing - non-communicable staph pseudomonas.

E. Potential liver problems (portal hypertension), especially with tetracycline and vitamin K synthesizing bacteria. Plan PT and PTT.

F. Decreased caries experience due to antibiotics.

G. Thicker saliva.

H. Check with patient re: chair position.

I. Dam with hole if breathing difficult.

J. Pancreatin replacement enzyme has been shown to inhibit plaque, lower caries incidence, and inhibit calculus formation.

K. Increased submandibular gland hypertrophy in 8% of studies. No real significance.

L. Esthetic considerations of tetracycline stained teeth. Vital bleach or laminates.

K. Sensitive taste buds?

X. PSYCHO-SOCIAL CONSIDERATIONS

A. Considerable emotional and financial stress of the family.

B. Need to be supportive and understanding.

C. Care must be exercised that C.F. person does not dominate the family environment, thus affecting treatment.

D. Parental guilt because of the genetic nature of the disease will further complicate the situation.

E. Adolescent will be unduly stressed by the circumstances and future of the disease.

F. If no contact has been made with the Cystic Fibrosis Foundation, such a suggestion by the dentist can provide the family with a new resource for environmental stability.

XI. ECONMIC CONSIDERATIONS

A. The monetary drain on the family can be considerable.

B. Dietary and dental needs can easily become secondary considerations to the family.

C. Education of the inter-relationship of the oral cavity, health and upper respiratory infections must be explained by the dentist.

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CONVULSIVE DISORDERS

I. DEFINITION/DESCRIPTION

A. Epilepsy-Greek word "to seize", "to lay on".

B. Epilepsy-not a disease; a nonspecific neurological clinical symptom of a pathological irritation of the central nervous system.

C. Seizure-an episode of cerebral dysfunction produced by abnormal, excessive, neuronal discharge occuring in the brain which may be caused by a large variety of conditions.

D. Two classifications: Acute or nonrecurrent and chronic or recurrent.

E. Acute convulsions-generalized tonic and clonic convulsions, similar to grand mal type of seizure.

F. Chronic convulsions-recurrent paroxysmic attacks during which time a person may become unconscious or partially unconscious as well as having tonic or clonic muscular spasms.

G. Chronic convulsions are different from acute convulsions-they occur more frequently and are in general of different etiology.

II. INCIDENCE

A. Most common during first two years of life.

B. Acute convulsions-six to eight per cent of all children do experience some type of febrile (acute) convulsion.

C. Febrile convulsions are most common type of acute convulsion.

D. Febrile convulsions generally occur between the ages of 6 months and 3 years.

E. In North America over half a million children annually have some type of acute convulsion; most common being a symptom of acute febrile illness.

F. Ninety five per cent of those involved- 6 months to 5 years of age at time of first convulsion.

G. Peak incidence is between 1 and 2 years.

H. One in five seem to have a family history of similar occurances.

I. No accurate information available on incidence in older age groups; evidence supports it is considerably higher in children than in adults.

III. ETIOLOGY

A. Acute (nonrecurrent) convulsions- result of fever, intracranial infections, intracranial hemmorrhage, toxic substances, anoxia, metabolic or nutritional abnormalities, acute cerebral edema and brain tumors.

B. Intracranial injuries of birth are single most common cause of convulsions in young infants.

C. Anoxia and hemorrhage are many times the result of the intracranial injury.

D. Most frequent causes of convulsions in late infancy and early childhood- acute infection, both intracranial and extracranial.

E. Idiopathic epilepsy- most outstanding cause of convulsions occuring midway through childhood.

F. Most febrile convulsions occur above 39 degrees centigrade.

G. Febrile convulsions- usually associated with infections involving upper respiratory tract.

H. Seems to be a familial tendency.

I. Majority of cases the cause of recurrent seizures can not be established; designated as idiopathic.

J. May be some specific genetic defect in cerebral metabolism, (in the recurrent form).

K. There are a variety of genetically determined conditions associated with seizures.

L. Etiology may be anatomic, biologic in nature or may be result of cerebral damage.

IV. PATHOLOGY

A. Common denominator- a structural or metabolic disturbance in the brain giving rise to seizure discharge.

B. Biochemical basis of seizure discharge remains incompletely understood.

C. Encephalographic tracings may show generalized abnormalities in idiopathic epilepsy.

D. Regardless of the etiology or pathology of the cerebral lesions, the pathophysiology of seizure discharge may be identical.

V. CLINICAL MANIFESTATIONS - General

General category on convulsive disorders are divided into subgroups to properly describe the different seizure activities:

A. Grand mall seizures.

1. May be preceded by a momentary "aura" of which very few can definitely describe.
2. A "motor aura" or localized twitching or spasm may also occur as a warning.
3. Other vague symptoms such as headache, irritability, intestinal disturbances, etc. may also forwarn patients and parents of the possibility of seizure activity.
4. These seizures are generalized convulsions.
5. Both tonic and clonic phases of muscular spasms are exhibited.
6. These spasms may occur simultaneously with a state of unconsciousness.
7. The patient may fall to the ground. Face will become pale, pupils dilate, eyeballs roll

upward or to one side, face may distort, and glottis may close. Head and limbs may be thrown about or be held rigid. Tongue biting may be associated with rapid opening and closing of the mouth during contraction of facial muscles.

8. Micturition and defication may result because of the force of contraction in the abdominal muscles.
9. Cyanosis due to head position and/or contraction of muscles affecting respiration.
10. Tonic phase generally lasts not more than 20-40 seconds.
11. Clonic phase will follow and can last for varying amounts of time.
12. Post convulsive period can be filled with an automatic, semi-dazed behavior. A generalized headache is not uncommon.

B. Petit mal seizures

1. Rarely seen before 3 years of age and usually disappear before puberty.
2. Girls are more often affected.
3. Seizure episodes consist of transient loss of consciousness similar to "dizzy spells", "absences" or "lapses".

4. Seizure may be accompanied by other minor motor movements such as rolling of the eyes.
5. Attacks usually last less than 30 seconds.
6. Frequency of seizures vary greatly from one to two per month to several (perhaps a hundred per day).
7. Prolonged episodes may occur and may resemble a mild grand mal episode.

C. Acute (febrile) convulsions
1. Generalized tonic and clonic, similar to grand mall type of seizure.
2. Most occur above 39 degrees centigrade.
3. Duration is usually less than a minute or two but may be more than ten minutes.

D. Miscellaneous - Other types of seizures also exists but to a lesser extent. These are the psychomotor seizure, focal seizure (Jacksonian), infantile myoclonic, myoclonic and akinetic seizures, nocturnal seizures and self induced seizures. The clinical manifestations of each of these will vary somewhat.

Manifestations - oral

A. General appearance- within normal limits.

B. Possibility exists that tooth development may have been affected morphologically. This morphologic variation could possibly be linked to an insult within the central

nervous system during tooth development. This variation could also be the result of a teratogen or metabolic type of disturbance occuring during the development period.

C. Gingival overgrowth secondary to phenytoin - can lead to:

1. Delayed exfoliation of primary teeth.
2. Delayed eruption of permanent teeth.
3. Mal-alignment of teeth and malocclusion.
4. Excessive accumulation of plaque leading to gingivitis.

D. Fractured and avulsed teeth.

VI. DIAGNOSIS

A. Medical history, physical examination, clinical course of disease all form a background for diagnosis.

B. In an infant or child when a convulsion lasts more then 20 minutes, it is very likely not a febile convulsion.

C. Proper laboratory tests should be included so as to rule out the possibility of systemic factors such as hypoglycemia, nephritis, lead poisoning, tetany, etc.

D. Main diagnostic finding- electroencephalogram in the identification of abnormal spike-wave phenomena.

E. Supplement to electroencephalogram is roentgenographic study used in determining the possibilities of abnormal characteristics within the skull such as intracranial

calcifications, increased densities or erosion of the base.

F. Computerized Tomography (CT Scan) is used for detecting location and nature of lesions within the cerebrum that could be responsible for recurrent seizures.

VII. TREATMENT

A. Aims of continuous therapy- to reduce the number of seizures, encourage the child to function at a natural level and to promote acceptance in community and home.

B. Drugs used for control of convulsions.

1. Phenobarbital and phenytoin (Dilantin) are the most commonly used for control of grand mal and psychomotor seizures.
2. Unless there is a specific contraindication administration of phenobarbital (3mg/kg/24 hrs.) in 2 or 3 divided doses to the child with grand mal, psychomotor, petit mal, infantile myoclonic, or mixed seizures is treatment of choice.
3. Phenytoin is the most common drug that is added to therapy in the case of grand mal, psychomotor and mixed seizures (2-3mg/kg/24 hrs. in 1 or 2 divided doses, up to a maximum of 300mg daily in the pediatric age range).

4. Zartontin (ethosuximide) is a common drug of choice, in addition to phenobarbital, in control of petit mal seizures. (250 mg/capsule, 6 capsule max./day).

5. Other medications for control of seizures might include valproic acid (Depakene), Mebaral, tridione (Trimethadione), mysoline (Primidone), diazepam (Valium), carbamazepine (Tegretal).

C. Another modality in treatment- diet therapy which revolves around the ketogenic diet, fasting, and restriction of fluid intake which in a majority of children tends to prevent epileptic seizures.

D. Emergency treatment- status epilepticus.

1. Status epilepticus- grand mal seizures keep occurring before patient fully recovers and therefore lengthens the duration of total seizure.

2. Drug treatment of choice consists of prompt I.V. administration of phenobarbital sodium.

3. Diazepam- administer undiluted 0.5ml./min up to a maximum of 6 ml. (30 mg.) usual doses is 5-10 mg.; within one minute the effect of the drug is usually apparent. Duration of 0.5- 3 hrs.

4. Diazepan is valuable because of prompt control of convulsion with the added benefit of

alleviation of anxiety in parents and assistants as well as the practitioner. It can however mask the underlying cause and therefore cause a delay in definitive therapy.

5. Phenobarbital- administer 60 mg. at 6 months of age up to 120 mg. at 2-3 yrs. or 5-6 mg./kg up to a maximum of 200 mg. If convulsion continues for over 15 minutes, then dosage may be repeated or if only partially controlled then half the initial dose can be administered.
6. Sedative therapy should be limited to single agent.

E. Emergency treatment- Recurrent convulsions:

1. During an attack the main objective should be to protect the patient from bodily injury.
2. Clothing about the neck should be loosened, head turned to the side so that pooled secretions are not aspirated and patient observed for signs of anoxia.
3. Oxygen administration is indicated in situations of prolonged convulsions where anoxia can become a problem.
4. Since most injuries to the tongue and other oral structures usually occur at the beginning of a seizure it is usually not of value to try

and prevent subsequent oral trauma by the use of bite sticks, spoons or other crude objects which could in fact cause additional damage.

5. Above all it is important to remain calm and especially during the post-convulsive recovery phase it will be important to be assurring to both the patient and parents. Elimination of unnecessary annoyances will help assure a calm atmosphere.

F. Emergency treatment- Febrile convulstions (acute):

1. Immediate management requires maintenance of airway, oxygen administration and ventilation as well as appropriate doses of anticonvulsant medication. An attempt should also be made to lower child's temperature.

2. Long term management may include daily administration of oral phenobarbital until child is out of age group when febrile convulsions are likely. This medication may be started after second or third seizure or may be given intermittently at discretion of the physician.

VIII. PROGNOSIS

A. Acute Convulsions.

1. Child with one febrile convulsion has a 30-50% chance of having another.
2. Younger patients (under 13 mo.) are more likely than older ones of having recurrent febrile convulsions.
3. There is a slightly greater chance that patients with a history of febrile convulsions will develop non-febrile convulsions-idiopathic epilepsy-then children in the general population.
4. Chances are also increased if convulsion has been of long duration (longer than 10 minutes).
5. There are also increased chances if patient has had more than one febrile convulsion.
6. Fifty per cent with 4 or more febrile convulsions will have subsequent non-febrile convulsions.
7. In general the outcome is good. Complete recovery is likely even with a long convulsion.

B. Chronic convulsions.

1. Depends upon any coexisting mental retardation, physical handicaps or possible organic disease and upon the adequacy of medical and environmental management.

2. Most evidence supports the thought that the usual convulsive episode does not cause irreversible damage.
3. Convulsions followed by permanent hemiplegia are probably more often the result of a vascular accident which occured before the seizure than to injury during it.
4. Grand mal seizures will tend to become more numerous unless the course is modified by proper therapy.
5. A number of patients with unquestioned idiopathic grand mal epilepsy appear to undergo spontaneous cessation of seizures after adequate treatment.
6. Epileptic patients who are otherwise normal seldom die or sustain serious injuries as a result of their disorder.
7. Patients who are well controlled medically rarely have seizures during participation in athletic activities.
8. With proper treatment most epileptic patients with normal mentality can be expected to maintain it.

IX. DENTAL CONSIDERATIONS

A. Adequate health history regarding previous seizures must be obtained.

B. Type of seizure must be clarified since 6-8% of all children have had one or more febrile convulsions.

C. Medications the patient is currently taking should be known by the practitioner.

D. The practitioner should know how well controlled the patient is and how frequently seizures do occur and their approximate duration.

E. Practitioners should be aware of what types of activities will elicit a seizure.

F. Always consult the patient's physician if questions regarding medication, seizure activity, etc. are left unanswered.

G. Dental patients should be advised to continue their usual daily drug therapy.

H. Sedation with alcohol should not be used if possible since alcohol in some epileptics can bring on a seizure.

I. Some restraint such as a Pedi-wrap or Papoose-board may be beneficial to protect an epileptic patient from personal injury as well as giving them a sense of security during appointment.

J. Staff should be trained to maintain the airway and keep the mouth as clear as possible during the seizure so that aspiration of secretions or a possible foreign body (clamp, bite block, matrix bands, etc.) are kept to a minimum.

K. In some instances it may be acceptable procedure to use a proper bite block or tongue blades to decrease tongue biting. This should only be attempted however if further insult will be prevented by this procedure and the procedure itself will not be traumatic.

L. Staff should be trained in emergency usage of proper medications and oxygen in treatment of status epilepticus.

M. Parents or guardians should be notified that a child has had a seizure with explanation about duration and seizure activity.

N. Should a seizure be of long duration- parent or guardian should be notified and information obtained as to previous seizures of this type and any other helpful information in controlling the seizure.

O. Important that staff remain cool, and collected both during and after the seizure and be reassuring to both the patient and parents.

X. PSYCHO-SOCIAL/ECONOMIC CONSIDERATIONS

A. Social adjustment of a child depends greatly on the degree of control of his/her seizures.

B. Attitude of child toward the disease generally reflects that of parents.

C. Psycho/social adjustment will also depend on how the doubts and fears of both parents and child are dealt with.

D. Realism and optimism are philosophies that will both have a long-term benefit.

E. Most children are encouraged to participate in regular activities.

F. Since seizures during athletic activities are rare in well controlled children, it may also be highly probable that they can compete in competitive sports.

G. For better adjustment of the child it is better to leave the discussion about discontinuation of medication until the child has been without seizures for a year. If treatment has been discontinued but needs to be resumed once again for better control, it will be emotionally difficult for both parents and child.

H. It is important to stress the need for independence during adolescence.

I. Child's teachers and psychologist as well as others involved (parents, physician etc.) should take a team approach to finding a realistic educational program.

J. Child with epilepsy wants to be "normal", to be independent, to be accepted and admired by peers. He/she will sometimes try to attain this acceptance by trying to achieve unrealistic goals.

K. They may sometimes test the fantasy that nothing is wrong with them and refuse to continue their medication and leave open the possibility of further seizure activity which could lead to an unnecessary set-back.

L. The patient who has had little opportunity to exercise judgement in daily activities is likely to use their handicap as a shield and barrier to normal involvement.

M. If a child gains excessive attention by having seizures, control by medication alone is likely to be difficult.

N. In most instances, avoidance of emphasis on the recurrence of seizures is helpful.

O. The patient needs an environment that will allow successful competition at his or her own level.

P. Because of the need for long term treatment using a multidisciplinary approach, the economic burden placed on the family may not only cause a financial hardship but may also cause resentment from other family members.

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MENTAL RETARDATION

I. DEFINITION/DESCRIPTION

A. Significantly subaverage general intellectual functioning existing concurrently with deficits in adaptive behavior and manifested during the development period. (AAMD)

1. I.Q. below 2 standard deviations from normal.
2. Impairment in adaptive behavior.
3. Manifestations of 1) and 2) before 18 years of age.

B. Primary causes of mental retardation may be considered to originate from biological factors which influence the biochemical and structural organization of the neural matrix or from experimental factors which influence the organization of function in the central nervous system during postnatal maturation or from varying combinations. (AMA)

1. Organic problem with
   a. Brain metabolism.
   b. Nervous system function.
2. Cultural deprivation.
3. Combination of 1) and 2).

II. INCIDENCE

A. Children

1. 2.3% of the population.

B. By age.

1. Less than 5.

a. Mentally retarded - 7%

2. 5-19

b. Mentally retarded - 72%

3. Over 19.

a. Mentally retarded - 21%.

C. Prevalence in U.S. by I.Q.

1. 0-20 - 87,500

2. 20-50 - 350,000 3% of the population

3. 50-70 - 5,276,755

D. Sex.

Males: females 3:2

III. ETIOLOGY

A. Organic cause - usually moderately-profoundly mentally retarded.

1. Pre-natal infections.

a. Rubella.

b. Syphillis.

c. Cytomegalic virus.

d. Toxoplasmosis.

2. Post-natal infections.

a. Meningitis.

b. Encephalitis.

3. Intoxications.

a. Maternal antibodies.

1. Rh incompatibility.

2. Erythroblastosis fetalis.

b. Drugs - pre-natal.

1. Alcohol.

2. Nicotine.

3. Heroin.

c. Lead poisoning.

1. 60-80 ug/100 ml - toxic levels.

2. Elevated levels - below 60 ug/100 ml. Significant incidence of mild and borderline M.R. in children.

4. Trauma and other physical agents.

a. Abnormal position in uterus.

b. Birth process.

1. Prenatal anoxia - Placenta detached prior to birth.

2. Internal hemorrhaging during birth.

c. Post natal trauma.

1. Battered child syndrone.

5. Disorder of metabolism or nutrition.

a. Inherited metabolic disorder.

1. PKU - Phenylalanine buildup.

2. Galactosemia - can't metabolize galactose.

3. Disorders of lipid metabolism.

4. Hurler's syndrome.

A. Error in mucopolysaccaride metabolism.

B. Normal at birth-gradual deterioration.

5. G-6-PD deficiency. Significant relationship with M.R. in caucasian males.

b. Endocrine disorders.

1. Hypothyroidism (Cretinism).

A. Treatment.

1. Thyroid hormone from animals.

2. 1st 6 months of life-90% normal IQ.

B. After 1st year - 2% normal IQ.

2. Pituitary

A. Deficiency of thyroid stimulating hormone.

B. Nephrogenic diabetes insipidis.

3. Hypothalamus disorder.

A. Cerebral gigantism.

B. Lawrence-Moon-Biedl syndrome.

4. Parathyroid disorder.

A. 1° Hyperparathyroidism.

B. Albright hereditary osteodystrophy.

c. Malnutrition.

1. Prenatal nutrition.

2. Postnatal nutrition - Younger malnutrition begins - greater effect on IQ.

6. Post-natal gross brain disease.

a. Tuberous sclerosis - progressive retardation.

b. Von Recklinghausen's disease - progressive retardation.

7. Unknown pre-natal influences.

a. Hydrocephaly - increased intracranial pressure.

b. Spina bifida - hydrocephaly.

c. Microcephaly - if 2 standard deviations below average for age and sex.

8. Chromosomal abnormalities.

a. Down's syndrome - 1/600.

b. Sex chromosome disorders.

1. Turner's syndrome.

2. Klinefelter's syndrome - 1/400 male births.

c. Genetic Inheritance of M.R. Syndromes.

1. Autosommal Dominant

A. Albright's Hereditary Osteodystrophy - Apert's Syndrome.

B. Myotonic Dystrophy

C. Basal Cell Nevus Syndrome

D. Reiger's Syndrome

E. Tuberous Sclerosis

F. Neurofibromatosis

G. Achondroplasia

2. Autosommal Recessive

A. Bird headed dwarf (Seckel's Syndrome)

B. Gangliosidosis

C. Lawrence Moon Biedl Syndrome

D. Marinesco-Sjorgren Syndrome

E. Familial microcephaly

F. Hurler's Syndrome

G. Sanfilippo Syndrome

H. Sjorgren-Larsson Syndrome

I. Riley-Day Syndrome

3. X-linked Recessive

A. Hyperuricemia

B. Hunter's Syndrome

C. Oral Facial Digital Syndrome

4. Chromosomal Abnormalities

A. Partial Deletion of Long Arm of Chromosome 18 (and 21)

B. Cri du Chat Syndrome

C. Down's Syndrome

9. Gestational disorders.

a. Prematurity

1. Abnormal position in uterus.

2. More likely for delivery problems.

3. Hyperbilirubinemia - 2% inadequate liver function.

b. More common in lower income-racial minorities.

B. No organic abnormality.

1. 33% of mentally retarded people.

2. Functionally retarded.

3. Interference with the development and differentiation of the CNS.

   a. Social deprivation.

   b. Lack of stimulation.

   c. Protein-calorie deficiency at a young age.

4. Usually only mildly mentally retarded.

IV. PATHOLOGY

A. M.R. not related to specific disease or syndrome.

1. Abnormal head size.

   a. Microencephaly.

   b. Megalencephaly.

2. Brain Deformities.

   a. Prosencephaly.

   b. Agenesis of Corpus Callosum

   c. Absence of Olfactory Nerves (Arbincephaly).

   d. Acrocephaly - abnormally round.

   e. Scaphocephaly - abnormally long.

3. Disorder of Cellular Migration.

   a. Microgyria.

   b. Pachygyria.

   c. Agyria.

4. Cortical Anomalies.

a. Brain warts.

1. Irregularly oriented nerve and glial cells.

2. Up to 3mm round plaques or nodules.

b. Gliotic encephalopathy.

1. Scarring of portions of the brain.

2. Common defect affecting mental function.

5. Atrophic neuronal changes.

a. Atrophy of neurons.

b. Atrophy of neuron's processes.

6. Eucephalomalacia

a. Rapid breakdown of brain tissue.

1. Secondary to severe damage.

2. Cavitation of affected portion.

b. Coalescence of vacules in rarified tissue.

B. Mental Retardation Secondary to disease.

1. Excessive gliosis (scarring).

a. Neurofibromatosis.

b. Tuberous sclerosis.

2. Cerebral cortical atrophy, scarring, encephalomalacia and multiple cysts.

a. Infection.

b. Epilepsy.

c. Local circulatory disturbance.

d. Asphyxia.

C. Somatic abnormalities associated with mental retardation syndromes.

1. Heart defects.

2. Hepatospleenomegaly.

3. Characteristic facies.

a. Flat profile.

b. Hypertelorism.

4. Increased incidence of cleft palate.

5. Abnormal head shape.

6. Skeletal deformity.

7. Skin disease.

8. Gonadal hypoplasia.

9. Decreased.

a. Body weight.

b. Arm length.

c. Foot length.

D. 12.1% have no detectable abnormality.

V. CLINICAL MANIFESTATIONS

A. General.

1. Moderate, profound M.R.

a. Retardation in appositional and endochondral growth.

b. Brachycephalic.

c. Microcephalic.

d. Dwarfism.

e. Failure to thrive.

f. Hypertelorism.

g. Skeletal ossification defects.

h. Skeletal aplasia.

i. Hirsutism.

j. Premature fusion of the cranial sutures.

k. Increased intracranial pressure-Hydrocephalus.

l. Diabetes insipidus.

m. Osteodystrophy.

n. Webbed neck.

2. Borderline - mild.

a. Many times not diagnosed.

b. Decreased intelligence by IQ testing only differentiation from normal population.

c. Most non-medical people won't know the person's intelligence is significantly less than normal.

B. Oral.

1. Mild- profound M.R.

a. Caries.

1. No difference versus normal population except:

A. Down's Syndrome.

1. Decreased caries prevalence.

2. Delayed eruption of teeth.

3. Delayed exfoliation of primary teeth.

B. Severe mental retardation.

1. Increase in dental disease because of lack of professional care, compromised diet and lack of daily oral hygiene.

b. Periodontal disease.

1. Higher than normal population.

c. Aberrant patterns of eruption.

d. Anomalies in the morphology of the dentition.

2. Oral components of syndromes of M.R.

a. Bifid tongue.

b. Delayed dentition or aplasia.

1. Albright's hereditary osteodystrophy.

2. Hypothyroidism.

3. Reiger syndrome.

c. Enamel or dentin hypoplasia.

1. Bird headed dwarf (Virchow-seckel dwarf)

2. Prader - Willi Syndrome.

3. Reiger Syndrome.

4. Sjogren Syndrome.

d. Gingival overgrowth.

1. Epilepsy - secondary to treatment with phenytoin.

2. Tuberous Sclerosis.

e. High palatal vault.

1. Cerebro-Hepato-Renal Syndrome.

2. Microcephaly.

3. Myototic Dystrophy.

4. Ring Rhromosome 18.

f. Hypoplastic Maxilla.

1. Alpert's Syndrome.

2. Crozon's Syndrome.

g. Macroglossia.

1. Down's Syndrome.

2. Hypothyroidism.

3. Kleebatschadel Syndrome.

4. Mucopolysaccharidosis.

h. Malocclusion.

1. Class II, Division I.

2. Anterior open bite.

3. 2° to retarded oral functioning.

i. Microdontia.

1. Down's Syndrome.

j. Micrognathia.

1. Bird headed dwarf (Virchow-Seckel dwarf).

2. Cri du Chat Syndrome.

3. De Lang Syndrome.

4. Prader Willi Syndrome.

5. Ring Chromosome 18.

k. Odontogenic Keratocyst.

1. Basal Cell Nevus Syndrome.

l. Overretained primary teeth.

1. Down's Syndrome.

m. Pegged shaped teeth.

1. Hurler's Syndrome (Mucopdysaccharidosis).

2. Cogenitial Syphyllis.

A. Mulberry Molars.

n. Port Wine Stain.

1. Sturge-Weber Syndrome.

o. Supernumerary teeth.

1. Down's Syndrome.

2. Oral-Facial-Digital Syndrome.

VI. DIAGNOSIS

A. Signs and Symptoms

1. Characteristic facies.

a. Ocular abnormalities - outgrowth of brain.

1. Micrognathia.

2. Cataracts.

3. Colaboma.

4. Retinal atrophy.

5. Mongolian or antimongolian slants of eyes.

6. Hypertelorism.

b. Premature fusion of the cranial sutures.

1. Protuberant frontal region.

2. Triangular frontal defect.

2. Dimished orienting reflex.

a. Decreased reaction to stimulus.

1. Weak to no reaction.

2. Decreased ability problem solving.

b. Extraneous stimulus reacts during problem solving tasks.

B. Laboratory tests.

1. Intelligence tests. - Average 100.

a. Wechsler Scale - Below 70.

b. Stanford Binet - Below 68.

1. Borderline 68-83.

2. Mild 52-67.

3. Moderate 36-51.

4. Severe 20-35.

5. Profound Below 20.

2. Hypothyroidism.

a. Destruction of thyroid.

b. Failure of thyroid stimulating hormone from pituitary.

3. Chromosome classification.

a. Karyogram.

b. Barr bodies - Buccal mucosa cells - Identify sex chromosomes.

4. Screening for PKU.

a. Phenylalanine in the urine-add $FeCl_2$-Green odor.

b. Phenylalanine in the blood.

1. Add blood to B-2 thinylalanine agar.

2. Add Bacillus subtilis to agar.

3. If growth of Bacillus subtilis-positive test for phenylalanine in blood.

VII. TREATMENT

A. Prevention.

1. Diagnosis of genetic malformation in utero.

a. Ultrasound.

b. Radiography.

c. Amniocentesis.

2. Metabolism disorder.

a. PKU - Prevent phenylalanine in diet.

3. Endocrine disorders.

a. Early diagnosis.

b. Genetic counselings.

c. Hypothyroidism.

1. Thyroid hormone from animals.

2. Given 1st 6 mo. of life - 90% normal IQ.

4. Rubella vaccine prior to pregnancy.

5. Pre-natal.

a. Adequate nutrition.

b. Elimination of infection.

c. No alcohol or drugs.

6. Early intervention of culturally disadvantaged children.

a. 1-2 years of age.

B. After diagnosis of mental retardation.

1. Develop behavior in a socially acceptable manner.

a. Behavior modification.

b. Speech therapy.

c. Non-vocal communication therapy.

d. Reading comprehension.

e. Psychotropic medications.

2. Improve the quality of life.

a. Optimal vision - corrective lenses.

1. Improved behavior.

2. Improved academic abilities.

b. Educational retarded - IQ - 50-79.

1. Repetition geared toward adjusting to everyday problems in life.

c. Trainable retarded IQ - 25-49.

1. Self help skills.

2 Social adjustment skills.

3. Economic usefulness in a sheltered environment.

VIII. PROGNOSIS

A. Mild - moderate M.R. - mental ability.

1. Plateau at a younger age versus normal.

2. Can rise during the 20 + 30's

B. Moderate profound M.R.

1. General decreased life span, especially if associated with a syndrome.

IX. DENTAL CONSIDERATIONS

A. Wide variation of behavior and level of understanding between mild and profound mental retardation. General considerations:

1. Know the patient's illness.

2. Treatment plan on an individual basis.

3. Reward good behavior and discourage inappropriateness.

4. Teach the parents or guardian the principles of preventive dentistry.

B. M.R. as part of a syndrome.

1. Congenital heart defects.

2. Ventricular - aortic shunts.

a. Complete medical history.

b. SBE prophylaxis.

C. Psychotropic drugs to control behavior.

1. Tricyclic anti-depressants.

a. Side effects with epinephrine - severe.

1. Hypotension.

2. Hypertension.

b. Xerostomia.

D. Mild-moderately retarded.

1. Explain new procedures slowly with repetition.

2. Introduce a minimum of new procedures.

3. Short appointments.

4. Oral hygiene can be improved.

a. Same procedures with normal intelligence except.

1. Need more appointments emphasizing oral hygiene.

2. Positive reinforcement.

E. Moderately - profound retarded.

1. Know the patient's communication level and understanding prior to bringing to operatory.

2. Ask parent, guardian or social worker.

a. Non-verbal cues.

b. Habits.

c. Detailed dental and behavorial history.

3. Problems with communicating with patient and vice versa.

a. Restraints and mouth props.

b. Acutely aware of physical discomfort as well as oral discomfort.

4. Stress the importance of preventive dentistry.

a. Sealants.

b. Fluoride.

c. Oral health.

F. Patient management - Because of the variability of behavior may require any of the below depending on the patients age, severity of M.R. and treatment needs.

1. Physical restraint.

a. Parent or auxillary - hold patient or keep patients hands out of treatment area.

b. Partial body - wrists or foot straps; trunk straps; either commercially available or home made.

c. Full body - commercially available eg: Papoose Board or Pedi-Wrap.

- Homemade eg. blanket or bedsheets with tape.

2. Positioning devices -

a. Bean bags.

b. Pillows to prop patient.

3. Sedation.

a. Route - dependent on patient, location and professional training.

b. Drug - dependant on patient needs, length of procedure and professional experience.

4. General anesthetic.

a. Patient selection dependant on treatment needs, severity of patient management and location for treatment.

b. Location - in hospital with medical management and complete O.R./dental facilities.

c. Risks vs benefits - must be fully understood before scheduling treatment.

X. PSYCHO-SOCIAL/ECONOMIC CONSIDERATIONS

A. Mild Mental Retardation.

1. Low income groups.

2. Predominately cultural deprivation.

B. Moderate - profound mental retardation.

1. All income groups.

2. Slightly more low income families.

C. Siblings of mentally retarded children.

1. Split between:

a. Ashamed and jealous because of parental attention.

b. Wanting to take cae of sibling.

1. Especially if retarded child is younger.

D. Parents.

1. Guilt feelings, especially if part of syndrome.

2. Problems with burnout.

a. Moderately to profound retarded.

b. Oral hygiene - low on the priority list.

E. Financial.

1. Many of the families are low income groups.

2. If associated with other medical problems, financial burden could be overwhelming to the family.

3. Dental treatment - can become a financial burden; therefore first appointment should be at an early age; comprehensive preventive program initiated immediately, more frequent recall appointments eg. 3 to 4 times a year and maximum use of preventive treatments to reduce dental problems in severely involved patients eg. SSC's instead of multiple surface amalgam restorations and use of sealants on occlusal surfaces.

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Table 3-1. Developmental characteristics of mentally retarded persons.

Degrees of mental retardation	Maturation and development (preschool age 0-5)	Training and education (school age 6-20)	Social and vocational adequacy (adult 21 and over)
Mild	Can develop social and communication skills; minimal retardation in sensorimotor areas: often not distinguished from normal until later age.	Can learn academic skills up to approximately sixth grade level by late teens. Can be guided toward social conformity. "Educable"	Can usually achieve social and vocational skills adequate to minimum self-support but may need guidance and assistance when under unusual social or economic stress.
Moderate	Can talk or learn to communicate; poor social awareness; fair motor development; profits from training in self-help; can be managed with moderate supervision.	Can profit from training in social and occupational skills; unlikely to progress beyond second grade level in academic subjects; may learn to travel alone in familiar places.	May achieve self-maintenance in unskilled or semi-skilled work under sheltered conditions; needs supervision and guidance when under mild social or economic stess.
Severe	Poor motor development; speech is minimal; generally unable to profit from training in self-help; little or no communication skills.	Can talk or learn to communicate; can be trained in elemental health habits, profits from systematic habit training.	May contribute partially to self-maintenance under complete supervision; can develop self-protection skills to a minimal useful level in controlled environment.
Profound	Gross retardation; minimal capacity for functioning in sensorimotor areas; needs nursing care.	Some motor development present; may respond to minimal or limited training in self-help.	Some motor and speech development; may achieve very limited self-care; needs nursing care.

From The President's Committee on Mental Retardation: The problem of mental retardation, DHEW Publication No. (OHD) 75-22003, Washington, D.C., 1975, U.S. Government Printing Office.

MUSCULAR DYSTROPHY

I. DEFINITION/DESCRIPTION

A. Muscular Dystrophy (MD) a progressive degeneration of striated muscles which has an hereditary basis.

B. One of the most common primary diseases of muscle.

C. Characterized by weakness and atrophy of skeletal muscles causing both disability and deformity as disease progresses.

D. Several clinical types are present and are classified on the basis of age of onset, distribution of weakness, sex incidence, hereditary pattern and clinical course.

E. Two most important clinical forms:

1. Pseudohypertropic type (Duchenne).
2. Facioscapulohumeral type.

II. INCIDENCE

A. MD makes up the largest single group of muscle diseases in children.

B. A high genetic mutation for MD. In 1 million people about 260 mutations per generation.

C. Occurs 3 times as often in males as in females.

D. Incidence of Duchenne MD.

1. X-linked recessive with a high mutation rate.
2. Risk is 1/2 for sons of carrier women; 1/2 of the daughters of carrier women are carriers.
3. 1/3 of the cases will be sporadic mutations.

4. Approximately 13 to 33 per 100,000 liveborn males are affected.

5. Prevalence is about 2.8 per 100,000.

E. Incidence of Facioscapulohumeral MD.

1. Transmission is usually as an autosomal dominant trait but, rarely is inherited as an autosomal recessive.

2. Occurance of partially-affected or abortive cases has been observed.

3. Prevalence is about 4 million livebirths.

III. ETIOLOGY

A. Theory - The main defect is the reaction of a muscle cell to injury. This may be due to a deficiency in a "maturation factor" which is normally stimulated by contact with the nervous system.

B. Theory - There is a fundamental biochemical defect present in the sarcoplasm or sarcolemmal membrane which limits continued growth and maintenance of the fibers.

C. Basic defect in unknown.

IV. PATHOLOGY

A. Muscle - a combination of regenerative and degenerative changes.

1. Early stage:

a. Variability in the diameter of individual fibers.

b. Forking or branching of thin fibers.

c. Vacuolation.

d. Hyalinization.

e. Atropic fibers arranged in a haphazard distribution.

f. Hypertropied fibers.

g. Small clusters of thin, basophilic staining fibers.

h. Fatty infiltration (lipomatosis)

i. Sarcolemmal nuclei - large, plump, rounded, vesicular with dark nucleoli.

2. Advanced stage:

a. Increased in interstitial connective tissue (fibrosis).

b. Sarcolemmal nuclei - clumps or chains of deeply staining bodies along the sheath.

c. Short, segmented fibers that have no tendinous connections.

3. End stage:

a. Disappearance of muscle fibers causing progressive paralysis.

b. Muscle reduced to fat and connective tissue.

B. Nervous system.

1. Central nervous system not significantly changed.

2. Peripheral nervous system.

a. Motor and plates are mildly atrophic.

b. Motor axons show an increase in terminal branching.

C. Cardiovascular system.

1. Myocardium composed of scar tissue.

2. Intima and media of blood vessels may be thickened and hypercellular.

V. CLINICAL MANIFESTATIONS

A. General

1. Duchenne MD.

a. Early onset - over 1/2 children manifest weakness by first 3 years of life.

b. Clumsiness in walking and tendency to fall.

c. Particular weakness of extensors and abductors.

d. Enlargement of gastronemius muscles.

e. Waddling gait with protuberant abdomen.

f. Lumbar lordosis due to weakness of spinal muscles.

g. Early loss of deep tendon reflexes in upper limb and knee jerk while ankle reflexes remain active.

h. Symmetrical involvement of muscles.

i. Contractures and skeletal deformities as result of muscle atrophy.

j. Mottling and cyanosis of leg skin.

k. Cramping of legs and abdomen.

l. Inability to raise arm above head.

m. Walks with feet apart and on the tips of toes.

n. Obese tendency.

o. Eventual wheelchair confinement.

p. Delayed puberty.

q. Mental retardation-borderline.

r. Persistent tachycardia.

s. Cardiomegaly in late stages.

t. Deterioration of respiratory function with severe carbon dioxide retention.

2. Facioscapulohumeral MD

a. Onset - between 6 to 20 years of age.

b. Initial muscle weakness of face and shoulder girdle muscles with later involvement of pelvic girdle.

c. Facial appearance - face is unlined and wrinkles are often missing from forehead and around eyes.

d. Inability to close eyes.

e. Elevation of scapulae on abduction of arms.

f. Some asymmetrical muscle weakness.

g. Difficulty in raising arms above shoulders.

h. Drooping or hunching forward of shoulders.

i. Difficulty in walking.

j. Slight speech impairment.

B. Oral

1. General type.

a. Inability to cough and clear accumulations of mucous from trachea.

b. Broad, flat, and flabby tongue.

c. Open mouth due to weakness of jaw muscles or to breathe more easily.

d. Vertical hyperplasia of dentoalveolar segment.

e. Tendency to drool.

2. Facioscapulohumeral type.

a. Inability to suck through a straw or whistle.

b. Lips and perioral area are often thickened due to pseudohypertrophy of orbicularis oris.

c. Pouting appearance of lips and a transverse smile.

VI. DIAGNOSIS

A. Symptoms.

1. Duchenne MD.

a. Weakness of hip girdle and later shoulder girdle.

b. Gowers' sign - weakness causes patient to rise from floor by climbing up on his own legs.

c. Weakness in one set of muscles with contractures in antagonist set.

d. Symptoms interpreted as child being lazy, clumsy and slow developmentally.

e. Pneumonitis - inability to clear respiratory secretions.

f. Myocardial failure - fibrotic and fatty infiltration.

2. Facioscapulohumeral MD.

a. Weakness begins in shoulder girdles.

b. A progressive change in facial appearance.

c. Cannot close eyes properly or whistle.

d. Speech becomes indistinct.

B. Tests.

1. Serum enzyme levels.

a. Creatine phosphokinase (CPK) - significantly elevated.

1. Noted in child before symptoms are detected.

2. Found in asymptomatic female carriers of Duchenne MD. 2/3 of all carriers have CPK values above normal range.

3. Measurement of this enzyme is most sensitive index for MD.

b. Aldolase - significantly elevated but not as sensitive diagnostically as CPK.

c. Phosphohexiosomerase, glutamic pyruvic transaminase, lacticode-hydrogenase are all increased.

d. Serum enzymes are much more elevated in Duchenne type than facioscapulohumeral form.

e. Enzyme activity is not proportional to degree of illness; it is highest at beginning of evolution when clinical state is good.

2. Urinalysis.

a. 3 major symptoms include hypocreatininuria, creatinuria and diminished tolerance to creatine.

3. In vitro studies - abnormal protein synthesis of muscle ribosomes.

4. Muscle biopsy studies - using electron microscopy, tissue cultures and electromyography.

5. Electrocardiogram - abnormal R and Q waves.

III. TREATMENT

A. Value of drug treatment has not been substantiated.

B. Antibiotics used to prevent serious complications from respiratory infection.

C. Fractures should be treated orthopedically.

D. Physical exercise especially swimming to prolong ambulation.

E. Muscles which are contracting should be stretched daily.

F. Tenotomy - questionable benefit.

G. Spinal support for scoliosis.

H. Braces for walking.

I. Wheelchair when person becomes nonambulatory.

J. Rigorous diet for weight control.

K. Diurectics for edema of the legs.

L. Must be kept from chilling because they become profoundly weak and stiff when cold.

VIII. PROGNOSIS

A. Muscular Dystrophy is not curable.

B. Duchenne type.

1. Rapidly progressive disease.
2. Nonambulatory within 10 years of onset.
3. Rapid deterioration if child is confined to bed due to illness for an extended period of time.
4. Most common cause of death - respiratory infection.
5. Death from inability to eat, respiratory infection or cardiac failure occurs usually in second decade.

C. Facioscapulohumeral type.

1. Slowly progressive disease with long stationary periods.
2. Generally benign disease in which majority of patients can have normal life expectancy.
3. Walking may become impossible in middle life.
4. Death due to respiratory infection may occur in middle life.

IX. DENTAL CONSIDERATIONS

A. Increase in total facial height with increased dentoalveolar height.

B. Mouth breather due to weakness of jaw muscles and poor respiratory function.

C. Protrusion of tongue which is broad, flat, and flabby and marked anterior openbite.

D. May have difficulty clearing pharynx due to weakness of pharyngeal musculature. Patient is unable to cough up any foreign material that may fall to the back of throat.

E. Tendency for patient to drool.

F. Tongue thrust causes outward inclination of incisors and splaying of teeth. Mouth guard may be appropriate to prvent dental trauma.

G. Poor general anesthesia risk because of impaired pulmonary function and large, flabby tongues.

H. Physical disability limits maintenance of oral hygiene.

I. Expanded maxillary and mandibular arches due to muscle weakness of cheeks and lips.

J. Lips and perioral area are often thickened due to pseudohypertrophy of orbicularis oris.

K. Protection necessary for eyes because they cannot close lids tightly.

L. Overeruption of first molars because of lowering of mandible.

M. High arch and narrow palate.

N. Correction of malocclusion using osteotomy and genioplasty.

O. Important to maintain body warmth because patient becomes very weak and stiff when cold.

X. PSYCHO-SOCIAL/ECONOMIC CONSIDERATIONS

A. Parents tend to be over-indulgent.

B. Child may be a whining invalid with hypochondriacal attitude.

C. Some children are quite depressed making them aggressive, frustrated and resentful.

D. Most children are well adjusted and accept their disability.

E. Children should be treated with continual encouragement and controlled optimism.

F. Children should be educated for some suitable sedentary occupation.

G. Care for these children is a financial burden due to hospitalizations and aids such as wheelchairs and braces.

H. Parental guilt is present because of the genetic component.

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SENSORY DISORDERS - BLINDNESS

I. DEFINITION/DESCRIPTION

Legally blind - central acuity does not exceed 20/200 in the better eye with correcting lenses (best correction) or whose visual acuity is greater than 20/200 but is accompanied by a limitation in the field of vision such that the widest diameter of the visual field subtends an angle of no greater than 20 degrees.

II. INCIDENCE

A. 10 to 15 million people in the world affected by total blindness.

B. Estimated 450,000 persons in U.S. are legally blind.

III. ETIOLOGY

A. Prenatal causes:

1. optic atrophy
2. microphthalmus
3. cataracts
4. colobomata
5. dermoid and other tumors
6. toxoplasmosis
7. cytomegalic inclusion disease
8. syphilis
9. rubella
10. tuberculus meningitis
11. developmental abnormalities of the orbit.

B. Postnatal causes:

1. trauma
2. retrolental fibroplasia
3. hypertension
4. prematurity
5. polycythemia vera
6. hemorrhagic disorders
7. leukemia
8. diabetes mellitus
9. glaucoma.

C. Optic atrophy and microphthalmus, frequently associated with brain damage.

D. Patients with optic atrophy and microphthalmus require frequent hospital visits and separation from families which may result in slow social development, making assessment of children's capacities difficult. These children are often represented as being backward or retarded.

E. Rubella can be associated with catastrophic impairment of the senses of vision, hearing, and other debilitating conditions of the fetus if infection occurs in early stages of development.

F. In general, if an effect is noted and one special sense, multiple system involvement is generally present.

G. Risk of fetal abnormality is greatest in first 16 weeks of pregnancy and to a lesser extent, in first trimester in general.

H. Retrolental fibroplasia is a condition associated with premature births (low birthweight) who have been exposed to high oxygen contents in incubators. Minute hemorrhages, which later produce scarring of the retina, take place. Since etiology is known it is now seen less frequently than it was in the past.

IV. PATHOLOGY

A. Because of the range and scope of the etiologies of blindness, the pathology involved in each will not be described.

V. CLINICAL MANIFESTATIONS

A. Decrease in visual acuity can be present at birth or may be a result of injury/infection later in life.

VI. DIAGNOSIS

A. Various diagnostic aids are employed in the diagnosis of disease process.

B. Such tests such as evaluation of visual acuity and visual fields, assessment of pupils, ocular motility and allignment, a general external examination, and ophthalmoscopic examination of the media and fundus should be a part of every pediatric evaluation.

C. When indicated, biomicroscopy (slit lamp examination), cycloplegic refraction, and tonometry may be performed by an ophthalmologist.

D. In certain special cases procedures such as ultrasonic examination, fluorescrin angiography, electrosentinograin (ERG), and visual evoked response (VER) tests are also indicated.

E. First clue of loss of vision in an infant may be nystagmus or strabismus.

F. Timidity, clumsiness, or behavoral change may be initial clues in very young.

G. Deterioration in school progress and disinterest in participation in school activities are common signs in older child.

H. Any evidence of loss of vision requires prompt and thorough ophthalmic evaluation. Many times this will require an extensive investigation involving neurologic evaluation, electrophysiologic tests, neuroradiologic procedures, and sometimes metabolic and genetic studies.

VII. TREATMENT

A. Treatments for the loss of visual acuity vary from surgery for partial correction to the wearing of special corrective lenses. There are also cases in which the patient will not benefit from either of these.

VIII. PROGNOSIS

A. Prognosis is generally poor although certain surgical procedures or corrective devices may aid in some correction of visual deficiency.

IX. DENTAL CONSIDERATIONS

A. There have been reports of relationships between the causes of serious impairment of the senses and anomalous development of teeth.

B. Dental anomalies associated with impairment of vision and probably closely linked to the primary systemic condition and course of disease process.

C. Importance of description of procedure and feeling of objects to be used as well as their sounds cannot be over emphasized with visually handicapped child.

D. Patients should be allowed to touch instruments.

E. Explanations should omit sight references as much as possible.

F. Inform patient adequately in advance of changes in chair position, etc. Touching patient during this change in position can be reasurring to patient.

G. The blind patient learns to become a good taster and mouther of objects and may reject certain tastes (i.e. toothpaste, topical anesthetic).

H. Inform patient of procedures that are going to occur (i.e. administration of local anesthetic), so they may not be "shocked" or suddenly scared by its administration.

I. Excessive description of procedures may make certain patients apprehensive.

J. Treatment must be highly individualistic, sensitive and responsive.

K. Prevention and continuity of care are of utmost importance to their care.

L. A team effort with dental staff, parents, counselors, physicians, etc. is needed to insure the maximum benefit to the patient of the entire dental experience.

X. PSYCHO-SOCIAL CONSIDERATIONS

A. Blind individuals have same basic needs for love and affection as the sighted.

B. Intellectual capacity and abilities in general will vary a great deal as it does in the normal population.

C. Demonstrated affection may play a larger role in life of a blind child.

D. Blind children should not have their functions performed for them. Encouragement toward development of self-help skills is important.

E. Touching and listening is the way in which these children learn.

F. Motor activity is important in their development because disruption of this motor activity tends to disturb development in other systems such as language and time and space perception.

G. Socialization is to be encouraged since outside stimulation tends to reduce activities that tend to be self-stimulating and egocentric such as rocking, head banging, finger flicking and eye pressing.

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SENSORY DISORDERS - THE DEAF

I. DEFINITION/DESCRIPTION

A. Mild hearing loss (15-30 decibel loss) (often called hard of hearing). Disability is slight, interferes little with development, and requires little help.

B. Partial hearing loss (30-65 decibel loss). Conversation is heard reasonably well. Amplification is usually required, combined with lip reading. Can often attend regular schools, although some special help may be necessary.

C. Severe hearing loss (65-95 decibel loss). Conversation must be very near and loud to be understood. Auditory and speech training efforts must be initiated early. Amplification may be helpful if supplemented by lip reading.

D. Profound hearing loss (95 decibel loss and above). Very few individuals have total hearing loss. Speech is not heard even with amplification. Early and intensive training in lip reading and other techniques such as finger spelling and signing is necessary. Some speech may be developed.

E. Classifications based on timing of insult:

1. the congenitally deaf born with a diability

2. the acquired or adventitiously deaf, born with normal hearing; hearing is diminished later in life.

F. Classification according to anatomic location of defect:

1. conductive loss, involving the apparatus of middle ear; conductive losses reduce the difference between air-conducted sounds and sounds produced by direct vibration of the skull (bone conduction).
2. sensorineural loss, involving the cochlea, cochlear nerves, or central pathways; speech discrimination is impaired.

II. INCIDENCE

A. Estimates vary with location, definition, etc.

B. Estimated 13,500,000 deaf and hearing impaired persons in the U.S. of which approximately 3 million are deaf; 400,000 lost their hearing before age 19 years.

C. Studies have indicated between 2-19/1000 have hearing defects.

III. ETIOLOGY

A. Prenatal causes:

1. heredity
2. infections
3. birth trauma

4. prematurity

5. blood incompatabilities

6. unknown causes (10%-20%)

B. Post natal causes:

1. infections

2. heredity

3. trauma

4. drugs

5. central deafness (cortical lesions)

C. Approximately 1/2 of childhood cases and 1/3 of adult cases fall into the category of hereditary or congenital defects. Generally, the disability is sensori-neural, the majority of cases (90%) being recessive in nature.

D. Hunter's syndrome, Waardenburg's syndrome, and osteosclerosis would be included under hereditary causes.

E. Osteosclerosis, usually autosomal dominant, a common cause of hearing loss in Caucasian adults, beginning in early life and progressing slowly.

F. Rubella infections account for approximately 20% of congenital deafness cases.

G. Studies report a marked increase in incidence with prematurity at birth.

H. Chronic serous otitis media- most common postnatal cause of conductive hearing loss in children.

I. Trauma to the temporal bone can produce hearing loss particularly if the bone is fractured transversely, thus producing inner ear damage and facial palsy.

J. Meniere's disease, which begins in middle life, affects both sexes equally and is characterized by hearing loss and vertigo.

K. Antibiotics such as streptomycin, neomycin, and vancomycin in sufficiently high concentrations are ototoxic.

IV. PATHOLOGY

A. Conductive losses result from interference in mechanical transmission of sound to inner ear.

B. Atresia, stenosis, and inflammation of external auditory canal; cerumen or foreign bodies in the canal; perforations of the tympanic membrane; congenital or acquired anomalies of the ossicular chain; and otitis media are among causes of conductive hearing loss. Most respond well to appropriate treatment.

C. Sensorineural losses result from destruction of or damage to cochlear mechanism or auditory nerve. This type of loss is almost always irreversible.

D. Mixed hearing losses result when a conductive loss is superimposed on a sensorineural loss. Typically conductive loss is temporary, often due to an upper respiratory infection, and lasts only until normal mechanical transmission of sound is restored.

V. CLINICAL MANIFESTATIONS

General

A. Hearing disorders at any age, even of a mild degree, can cause problems of speech, language, and learning.

B. Hearing loss is usually described as mild to profound.

C. Many areas of development are affected when there is a serious hearing impairment.

Oral Manifestations

A. Impairment of the senses, such as deafness, combined with dental defects have been reported with such conditions and syndromes as the ectodermal dysplasias and oculodentodigital (ODD) dysplasia.

B. Deafness can also be associated with rubella and prematurity, both of which have an increased incidence of enamel dysplasias.

C. Bruxism is also encountered more frequently in the deaf population although the reason for this is not known. It may serve as a form of self-stimulation.

VI. DIAGNOSIS

A. Measurement of auditory thresholds is not enough to define special needs of a child with impaired hearing.

B. Important to know also whether the loss is unilateral or bilateral, sensori-neural, conductive, mixed, and whether it is progressive.

C. Important to note behavioral characteristics such as child's visual attentiveness, ability to relate to others, communicative style and intent, vocal quality, distractability and relationship with parents.

D. Hearing evaluation consists of various tests to determine thresholds of sensitivity to pare tones presented to the ear by air and bone conduction.

E. Pure tone testing is accompanied by examination of child's threshold for speech reception and discrimination.

F. Hearing acuity of younger children is apt to be expressed in terms of their hearing awareness threshold or speech awareness threshold.

G. Impedence audiometry used to assess the integrity and function of peripheral hearing mechanism. Evaluation consists of such things as tympanometry, static compliance, and acoustic reflex thresholds.

H. Central auditory testing, which attempts to discriminate between peripheral and central dysfunction in child with auditory impairment, is in its infancy.

I. Other tests in speech as well as evoked response audiometry techniques are also methods of hearing evaluation being employed.

VII. TREATMENT

A. Most conductive losses respond well to treatment.

B. Early treatment is important to avoid possibility of sensorineural loss due to toxic materials passing into inner ear.

C. Sensorineural losses are almost always irreversible.

D. Treatment of hearing impairments may include surgery, other forms of therapy, preventive measures as well placement of hearing aids.

E. Special education and counseling of both parent and child may also be deemed necessary.

VIII. DENTAL CONSIDERATIONS

A. Deaf persons need to see what the dental team is doing.

B. Use of visual explanation of procedures is important; they do not like experience they cannot see.

C. The "tell" portion of "tell-show-do" must be both a visual and tactile experience for deaf patients.

D. Many times use of a picture or drawing may be useful in conveying an idea. Written information may also be valuable in an explanation of procedures.

E. Sometimes it is helpful to establish "signs" the patient may use during the procedure to indicate such things as discomfort that they may be unable to express verbally.

IX. PSYCHO/SOCIAL CONSIDERATIONS

A. Paramount concern in the development of children with severe to profound hearing loss is their impaired ability to learn verbal communication and language.

B. Methods of total communication using sound amplification, sign language, and speech to help the child learn communication and language have enabled children to develop more wholesome, normal relationships with their families and others in society.

C. The impairment of hearing affects many areas of development of other senses and activities therefore may be parental rejection of child.

D. Isolation is both an enforced and voluntary aspect of severe hearing impairment. Child may experience forced isolation because of an inability to communicate.

E. They have a desire to imitate. When desire and motivations are frustrated, the response may be defensiveness or withdrawl, usually the latter.

F. Since communication is slowed, deaf children are usually late in acquiring self-help skills and social adjustment.

G. Self-perception may be a problem that will be difficult to overcome.

H. They may receive inadequate schooling and training and as a result are placed in jobs below their potential, resulting in an even lower self-perception.

I. Parents may feel guilt, shock or disbelief and will have many questions regarding treatment, outcome, etc.

J. Parents must learn to accept the child and limitations of the disability and be willing to become involved in child's therapy in order for the child to become more fully socialized and adapted to this disability.

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PERIODONTAL DISEASE IN MENTAL RETARDATION

M. Michael Cohen, Sr., D.M.D.

Introduction

The occurrence and control of periodontal disease in mental retardation presents a unique challenge to dentistry. The prevalence of periodontal disease in the severe and profoundly mentally subnormal individual presents a special challenge to the dental practitioner and dentists who have had special training in dental care for the mentally retarded. In many instances because of their intellectual impairment and need of custodial care, it is essentially impossible to institute a program of preventive dentistry. However, it is possible to integrate a positive preventive program in moderate and mildly mental subnormal individuals. A plaque control program, proper diet and routine prophylaxis and scaling may be evolved. Recall reinforcement prevention utilizing deep scaling, root plaining, occlusal adjustment, surgical procedures (gingivoplasty, gingivectomy, flap surgery) is a necessity to maintain oral health. Dietary reinforcement and satisfactory restorative dentistry should be the goal of the practitioner for the maintenance of a healthy periodontium.

It may be assumed that periodontal disease in mentally retarded individuals is more common than in normal individuals (Figures 1, 2, 3, 4). In the majority of cases there is a relationship between the occurrence of periodontal disease, mental and developmental characteristics and many other factors.

Recently there has been an unusual interest in the nature, diagnosis of formation and pathological implication of dental plaque in periodontal disease. Plaque may be described as microbial masses, gel-like mats, microcolonies, microcosms, microbiotas zooglia or

communities of microorganisms closely adherent to the tooth or restoration. Plaque is not stain, pellicles materia alba or debris. Plaque may be visualized by a disclosing solution since most plaque is colorless.

Stains on the teeth are extrinsic in nature and are products of chromogenic bateria, food conbinations, tobacco, medications and any other material that may enter the oral cavity. Pellicles may be developmental (Naysmyth's membrane) or acquired. The acquired pellicle is a bacterial free film of glycoprotein and may be lipid derived from the saliva and/or gingival fluid and may cover the entire tooth surface. It often becomes stained any may in some areas become colonized by bacteria. When covered by bacteria it becomes part of the plaque.

Materia alba is a loosely adherent grayish white to yellow mass of bacteria and cellular debris which involves the plaque principally along the gingival margin. It is unorganized and a product of mechanical accumulation.

Debris is principally food particles which adhere to the gingival tissue and it is not part of the plaque. When it becomes impacted and broken down by enzyme action, it may constitute to soluble material for metabolic activity within the plaque.

For many decades it has become clear that a vital factor in periodontal disease is an accumulation of a complex of microorganisms. As research progresses it has become apparent that there appears to be specific sites for specific microorganisms to colonize. It is not the

intention of the author to make a precise judgement as to which site and combination of microorganisms are responsible for the specific periodontal lesion under consideration. It becomes apparent that investigators have an unusual opportunity to study periodontal disease in the mentally subnormal individual since many, because of their intellectual impairment, and many other factors, cannot practice oral physiotherapy and benefit from routine dental care and prophylaxis. It is essentially a virgin territory for investigators to observe the progress of periodontal disease without benefits of routine oral hygiene, periodontal treatment, the benefits of preventive procedures that are essential for the maintenance of a healthy periodontium. However, it is possible to integrate a positive preventive program in moderate and mildly mental subnormal individuals. A plaque control program, proper diet and routine prophylaxis and scaling may be involved. Recall reinforcement prevention, utilizing deep scaling, root planing, occlusal adjustment, surgical procedures (gingivoplasty, gingivectomy, flap surgery), is a necessity to maintain oral health. Dietary reinforcement and satisfactory restorative dentistry should also be the goal of the practitioner for the maintenance of a healthy periodontium.

Bacterial Flora of the Oral Cavity

The mouth, because it is warm and moist, creates an environment where bacteria thrive and colozize - large numbers of bacteria are indigenous to the oral cavity and are anerobic in nature and making it

more difficult to isolate and study them than the aerobic or facultative forms. Individuals with mental retardation, particularly the moderate to severe and profound types, frequently contaminate their mouths with foods, fingers or feces. Almost any organism may be isolated from the oral cavity at any given time.

Indigenous oral bacteria have a pathogenic potential and are etiologic agents to a variety of infections that include dental caries, periodontal disease, actinomycosis, subacute bacterial endocarditis and mixed anaerobic forms affecting many body tissues.

Indigenous organisms may be found in the mouth of all individuals and they are abundant.

Transient contaminants are found only in a small percentage of the population and when found their numbers invariably are low. A definitive study of the oral flora in the population of the mentally retarded individual is presently not available.

When such a study is available it is possible that we may have a better understanding of the etiologic factors involved in periodontal disease prevalent in the population of the individual with mental retardation. Treatment is invariably more successful when we know the etiologic factors.

It will then be possible to standardize examinations and determine the role of the host, the site of the periodontal involvement and the microorganism involved. It will also be possible to compare developmental characteristics and the degree of mental retardation in institutionalized and non-institutionalized individuals.

When standards of examination are established this will clarify the understanding of the prevalence of periodontal disease in the various types of mentally retarded individuals.

Recently, Tesini has stated that a higher prevalence of periodontal disease with a low prevalence of dental caries appears to be consistent finding.

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12. Socransky, SS, Manganiello AD: The oral microbiota of man from birth to senility. J Periodontol 42:485-496, 1971.

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Gingivitis

Studies have demonstrated that gingivitis correlates with large accumulations of bacteria in the gingival crevice on the teeth adjacent to the gingival margin. Leo and his coworkers have shown that gingivitis invariably develops from 10 to 21 days following the cessation of oral hygiene measures. When oral hygiene is resumed the gingiva returns to a healthy state in a short period of time.

Gingivitis is a constant finding in the severe and profound non-institutionalized mentally retarded individual since it is difficult to carry out routine oral hygiene measures daily and the problem is compounded in making available regular dental visits for routine oral prophylaxis. Parents or guardians must be counselled and taught methods to debride the mouth and gingiva of plaque and debris that accumulates. The following are suggestions on methods for debriding the oral cavity in the severe and profound mentally retarded non-institutionalized individual:

1. Electric toothbrush operated by the parent or guardian. One or two brushings daily will help maintain a reasonable degree of mouth cleanliness.
2. A water syringe with equal parts of hydrogen peroxide (3%) and water rinsed twice daily.
3. A wet 2 x 2 sponge may be used to wipe all surfaces of the oral cavity (tongue, cheek, gingiva, teeth). The sponge may be dipped into a solution of salt water (1/4 teaspoonful) and sodium bicarbonate (full teaspoon) in 4-6 ounces of water.

The patient should be seen by a dentist (one trained in the care of the handicapped) at six month intervals for routine oral prophylaxis and fluoride treatment, if possible.

Medication and anesthesia may be necessary for the office and clinic procedure.

If daily and routine oral physiotherapy is not instituted, chronic destructive periodontal disease results with pain, infection, and early loss of teeth.

Gingivitis

Suggested Readings

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SEIZURE DISORDERS (DILANTIN)

In mental retardation, because of brain damage, trauma and a combination of other factors, the patient manifests various forms of seizures, which are accompanied by loss of consciousness and a host of other symptoms. The most effective and widely used anti-convulsant is Diphenylhydantoin (Dilantin).

Dilantin prescribed for extended periods of time for the treatment of epilepsy may produce the follwing side effects and idiosyncratic reactions: Nystagmus, blurred vision, slurred speech and ataxia, which invariably subside following drug withdrawal. Skin rashes have been reported in 5 to 10% of patients as well as a rough appearance of the skin. Depression of one or more parameters of the cellular and/or humoral immune response have been reported occurring in up to 60%. Occasionally aplastic anemia and the Steven-Johnson syndrome may occur. Fibrous hyperplasia of the gigiva may occur in up to 57% of the patients on Dilantin therapy less than 26 years of age. In approximately 20-25% of patients, this drug induced effect is reversible upon drug withdrawal. Thickening of the skull and coarseness of facial features have been observed in long term anti-convulsive therapy.

Diphenylhydantoin (DPH Dilantin)

Suggested Readings

DILANTIN

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2. Gardner, A.F., Gross, S.G. and Wayne, L.E.: An investigation of gingival hyperplasia resulting from diphenylhydantoin in sodium therapy in seventy-seven mentally retarded patients. Exp. Med. Surg. 20:133, 1962.

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GINGIVAL HYPERPLASIA ASSOCIATED WITH DILANTIN THERAPY

Gingival hyperplasia in the mentally retarded is common and there are two types: non-inflammatory gingival hyperplasia and inflammatory gingival hyperplasia. Gingival hyperplasia may occur early (several weeks or in several months after the beginning of Dilantin therapy). Dilantin gingival hyperplasia is a serious problem that occurs particularly in the individuals that are moderately or profoundly retarded, and who have gross enlargement with superimposed inflammation due to a lack of oral hygiene. The gingiva become loose and edematous, bleed readily and create a serious problem for the parents, providing the patient is homebound, and likewise the institutionalized in maintaining any form of oral hygiene.

The hyperplasia begins as a bead-like facial or lingual enlargement. As the hyperplasia progresses, the marginal and papillary

enlargements unite. Often the tissue becomes massive and may involve part or all of the crown and, in many instances, cover the entire tooth. Dilantin hyperplasia may occur in mouths where local irritants are abundant. Local factors which include plaque, materia alba, poor restorations and food impaction usually enhance gingival hyperplasia. The gingival hyperplasia is found to be more marked in the anterior region of the mouth (Figure 5). The hyperplasia occurs in the following order of severity: Maxillary anterior facial, mandibular anterior facial, maxillary posterior facial and mandibular posterior facial (Figures 6, 7, and 8). There appears to be no satisfactory treatment for the gingival hyperplasia. Replacement of the drug with another anti-convulsive drug followed by surgical intervention appears to be the most satisfactory treatment.

Pressure appliances have been recommended following surgical removal of hyperplastic tissue. The appliance is fabricated like a tooth positioner and is adapted firmly to the gingival tissue. The most important aspect of this treatment is meticulous oral hygiene (oral physiotherapy). The appliance may also be used when there is mild hyperplasia, when surgery is not indicated. The best results are obtained when the appliance is worn at least three or four hours daily, and meticulous oral physiotherapy is practiced. (Figures 9-17)*.

*Courtesy of Manuel M. Album, D.D.S., Jenkintown, Pennsylvania.

PEROIDONTAL DISEASE IN DOWN'S SYNDROME

Numerous reports have confirmed that periodontal disease is a prevalent finding in patients with Down's Syndrome. (Figure 18).

More than three hundred clinical signs have been described in children with Down's syndrome: cardiac disease in the neonatal period, hematological abnormalities, occular signs (Brushfields spots), seizure disorders, congenital heart disease and a host of other abnormalities have been noted. Down's individuals are prone to systemic neutrophilic deficiencies which makes them more susceptible to leukemia and other diseases. The role of neutrophilic deficiencies - the high incidence of periodontosis in Down's Syndrome is interesting and requires more study and clarification.

When individuals have abnormalities of organ systems, it may be expected that the periodontium, the oral flora, salivary glands, as well as other anatomical structures in and about the mouth are affected and deficiency abnormalities results.

Reports by Cohen emphasized severe periodontal disease in children with Down's Syndrome. In one study Cohen et al found that 96% of 100 institutionalized patients with Down's syndrome had periodontal disease. Only 6% of control dental clinical patients of comparable age had periodontal disease. Marginal gingivitis, alveolar bone loss with loosening and exfoliation of teeth, especially lower central incisors were observed (Figure 19). Materia alba and supragingival and subgingival calculus were also noted (Figures 20, 21, 22). Superimposed necrotizing ulcerative gingivitis was found in 29% of the patients.

Brown and Cunningham reported periodontal disease in 90% of patients with Down's syndrome. Over 70% of those under six years of age had marked alveolar bone loss. In older patients, severe alveolar bone loss and gingival recession were observed. In some cases, alveolar bone loss and concomitant loosening of teeth preceded the onset of gingivitis (Figure 23).

Keyes, et al commenting on a clinical and microbiologic survey of children and young adults with Down's syndrome, suggested that these patients react severely to bacterial infection on surfaces of their teeth, which spreads along the root surfaces and infects the alveolar bone resulting in destructive periodontal disease. Bacteriologic studies of stained smears of deposits removed from tooth surfaces revealed that gram positive cocci rods, and other forms were especially prevalent. Certain strains of actinomyces which were isolated from some of these patients have caused periodontal lesions and root caries in experimental animals.

The oral structures may be affected by the following local factors: 1. Lack of dental care. 2. Tooth migration. 3. Occlusal disharmonies (Figure 24). 4. Tooth agenesis. 5. Mouth breathing. 6. Bruxism. 7. Excessive oral habits. 8. Tongue thrusting. 9. Finger and thumb sucking. 10. Lack of masticating food properly. 11. Lack of routine and regular dental care and lack of supervised oral hygiene and oral physiotherapy and bone loss (Figures 25 and 26). When all of the above factors are considered together with the mental, social, and educational factors superimposed, the complexity of the problem of the craniofacial, orofacial, and periodontal involvement in Down's Syndrome is apparent.

DOWN'S SYNDROME

Suggested Readings

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Figure 1: Seventeen year old profoundly retarded male, non-institutionalized. Note generalized periodontal involvement due to lack of dental care and oral physiotherapy.

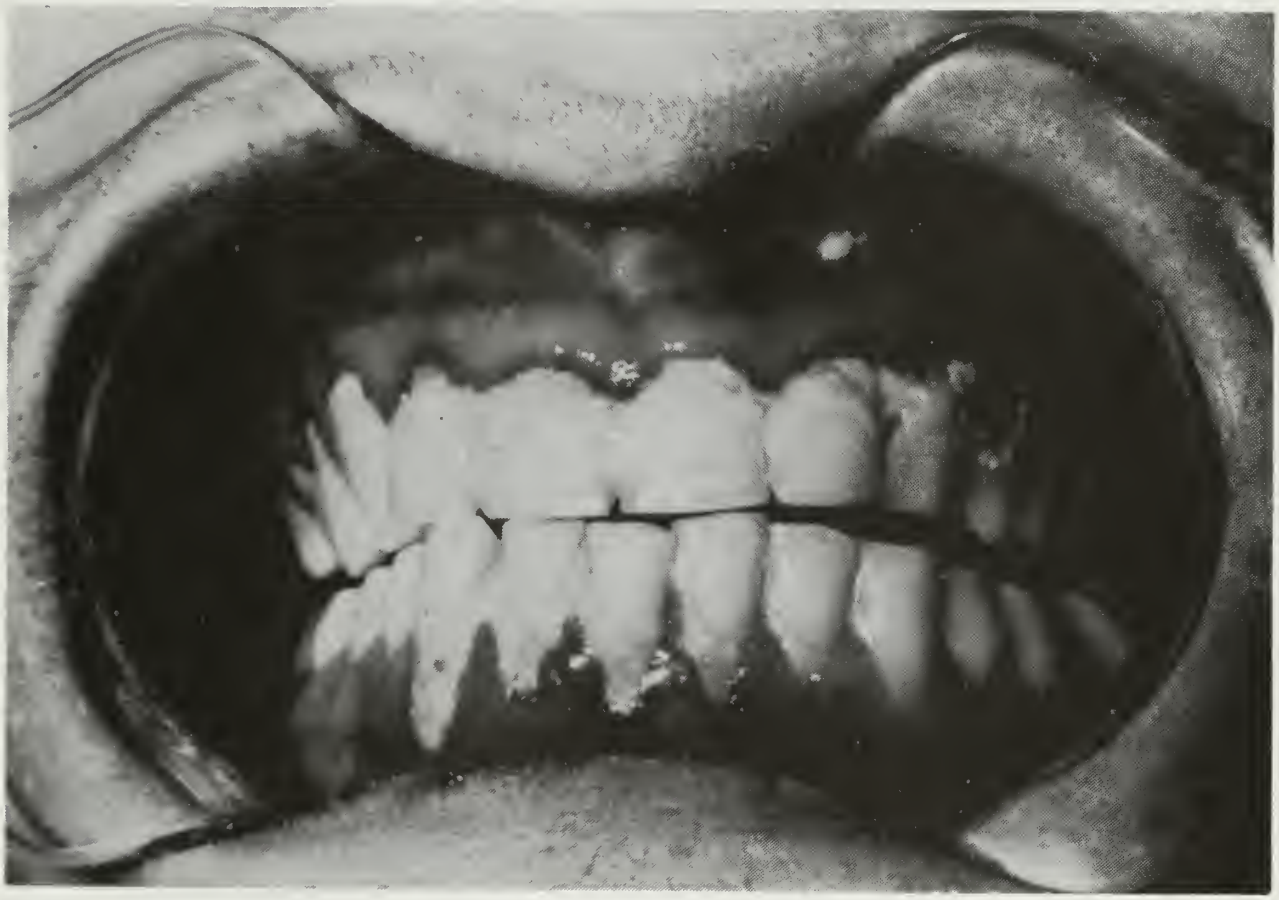


Figure 2: Sixteen year old profoundly retarded male, non-institutionalized. Note occlusal disharmony and generalized periodontal involvement, due to lack of dental care and oral physiotherapy.

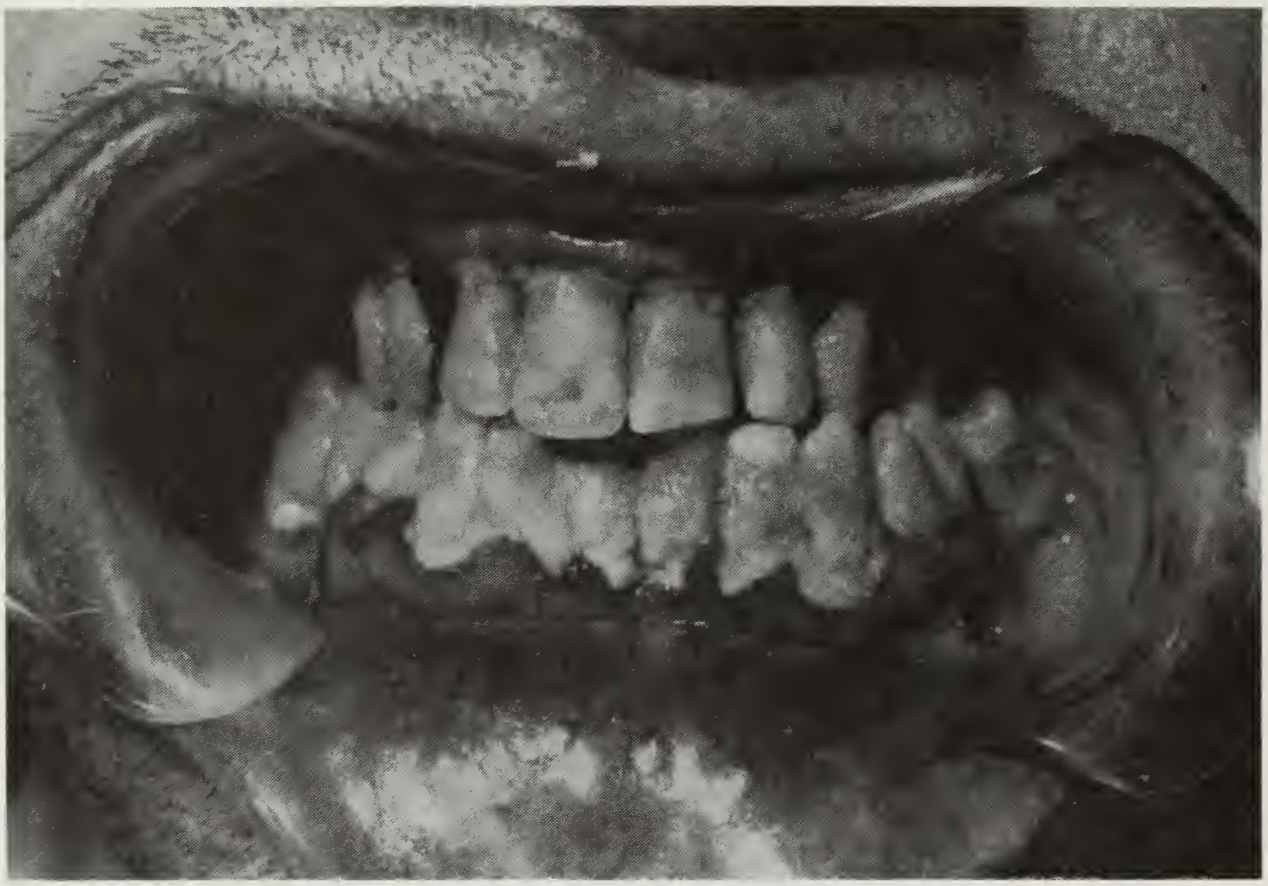


Figure 3: Ten year old severely retarded male, non-institutionalized. Note proliferative gingival enlargement on maxillary anterior regions and chronic necrotizing gingivitis in mandibular incisor regions.

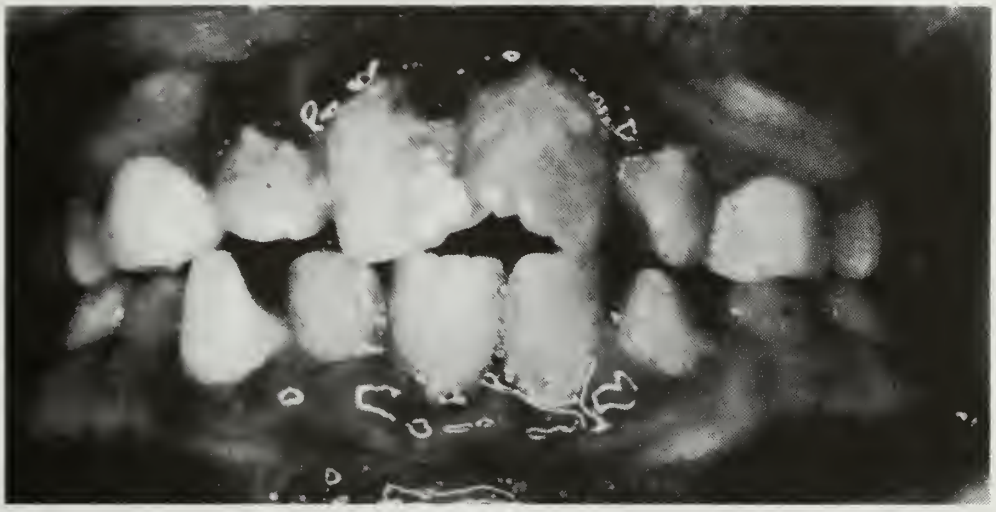


Figure 4: Twenty-two year old profoundly retarded male, non-institutionalized. Note bilateral crossbite and chronic destructive periodontal disease.

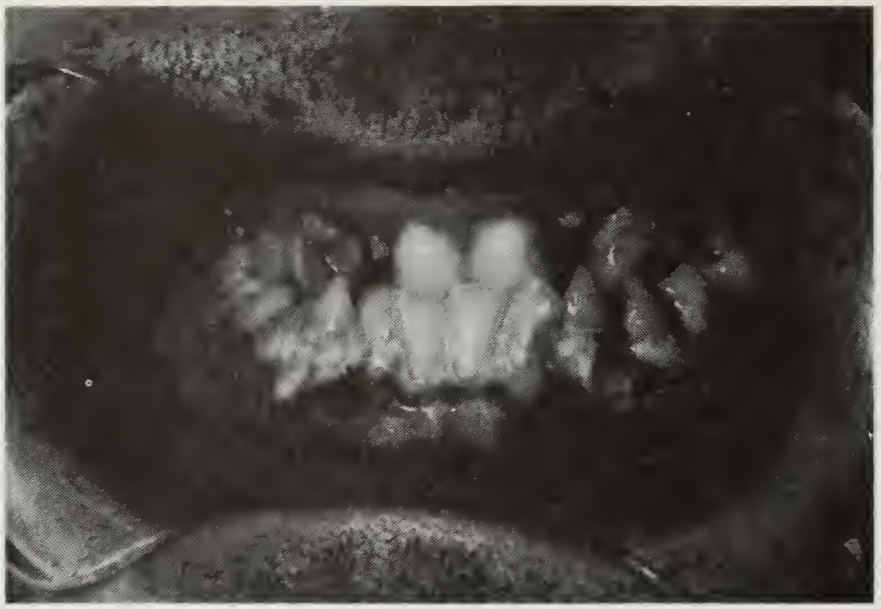


Figure 5: Severely retarded individual, non-institutionalized, with seizure disorders, on Dilantin therapy. Note generalized gingival hyperplasia.

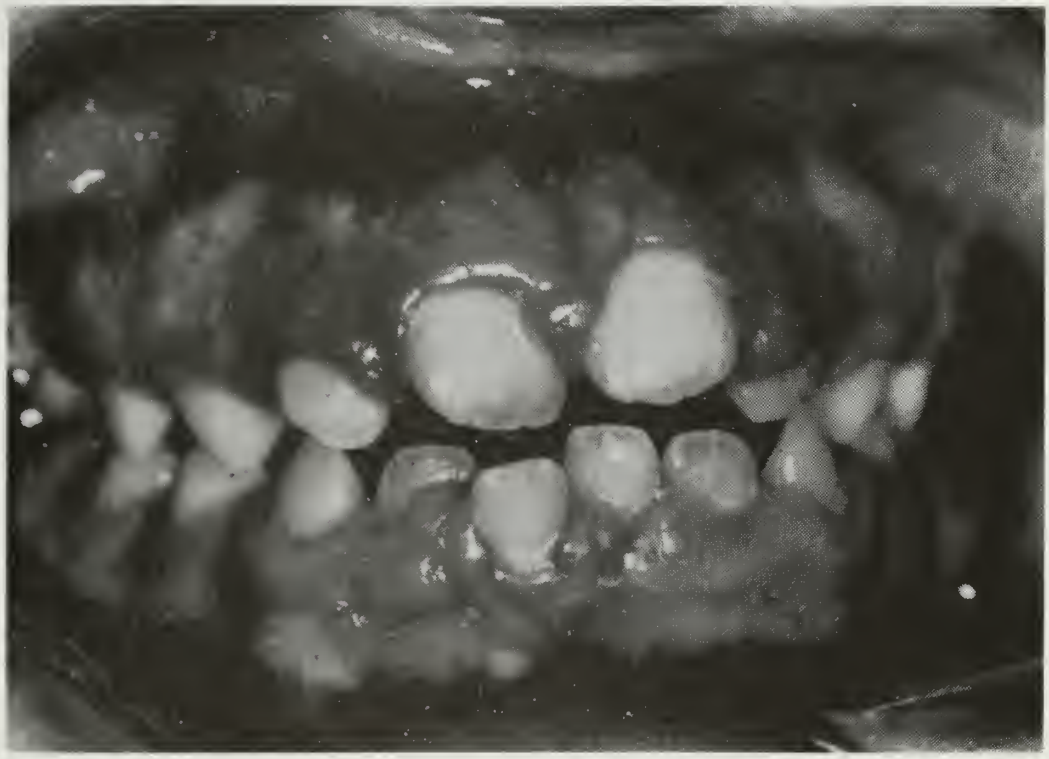


Figure 6: Profoundly retarded individual, non-institutionalized, with Dilantin disorder. Note proliferative generalized gingival hyperplasia.

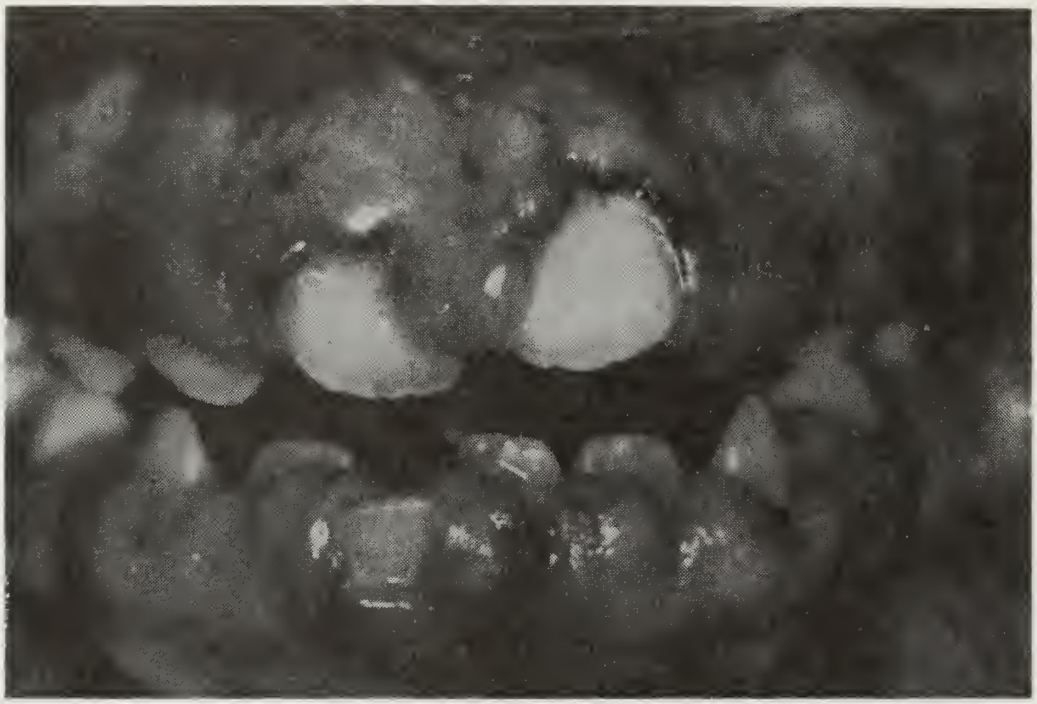


Figure 7: Profoundly retarded individual, non-institutionalized, with gingival hyperplasia. Note proliferated hyperplasia involving mandibular teeth. Maxillary teeth were absent with no hyperplasia on maxilla.

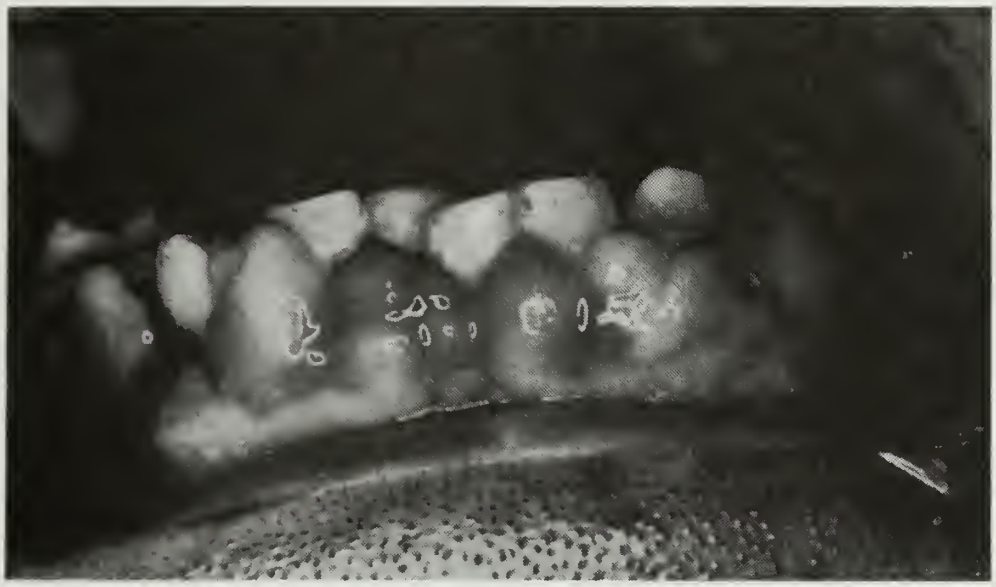


Figure 8: Profoundly retarded non-institutionalized individual on Dilantin for a long period of time. Note generalized gingival hyperplasia throughout the mouth interferring with the eruption of teeth.

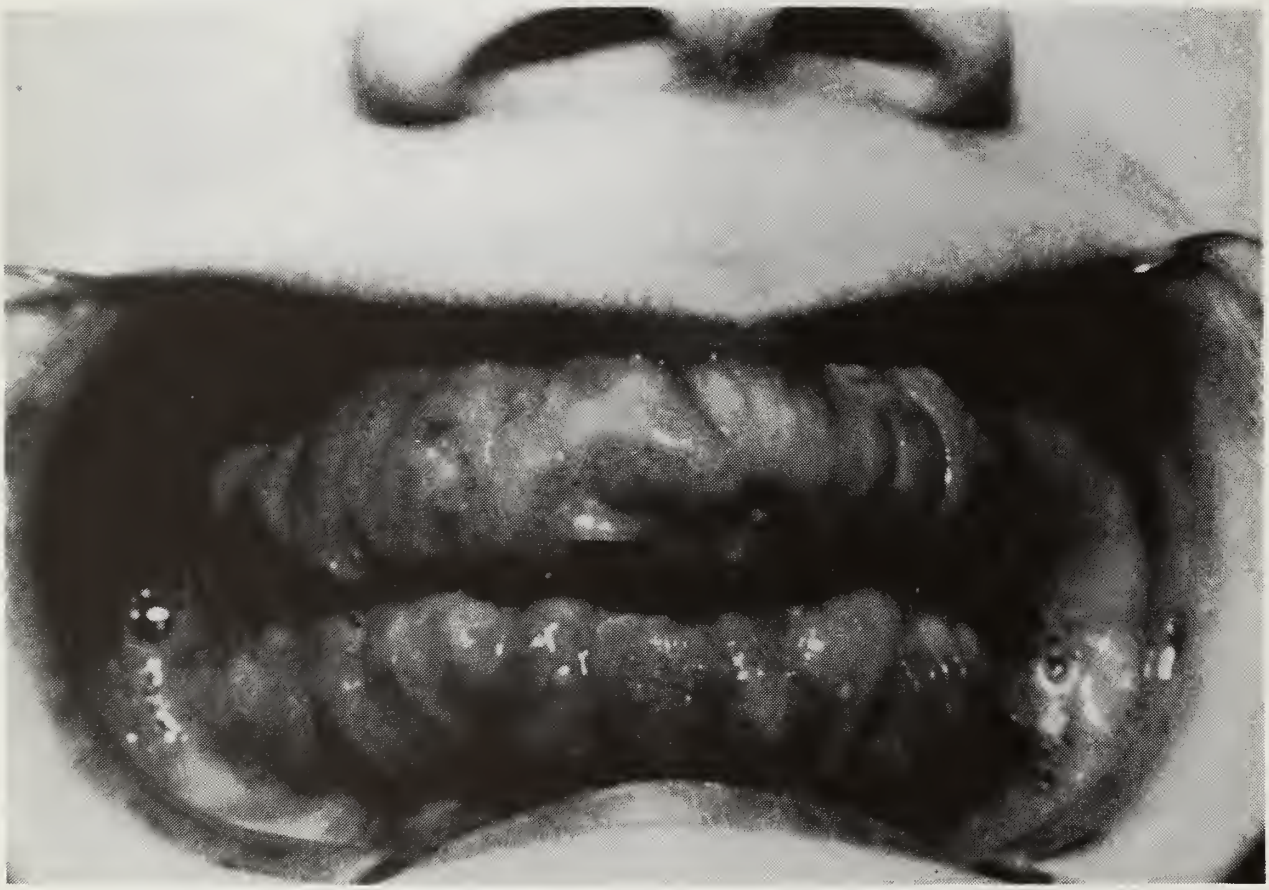


Figure 9: Patient receiving Dilantin Therapy. Note extensive hyperplasia of gingival tissues. Also movement to teeth caused by excessive gingival tissues.

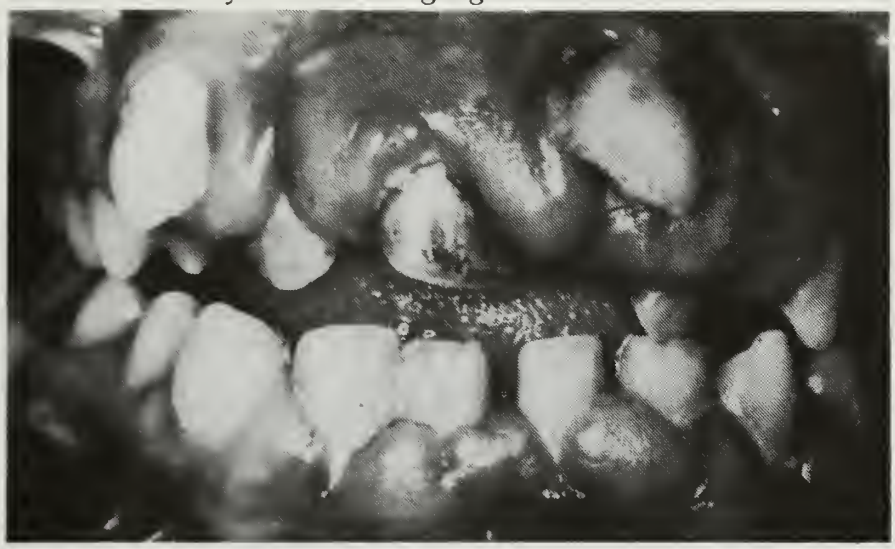


Figure 10: Prophylaxis and Scaling prior to gingivectomy. This permits clearer picture of gingival tissue.

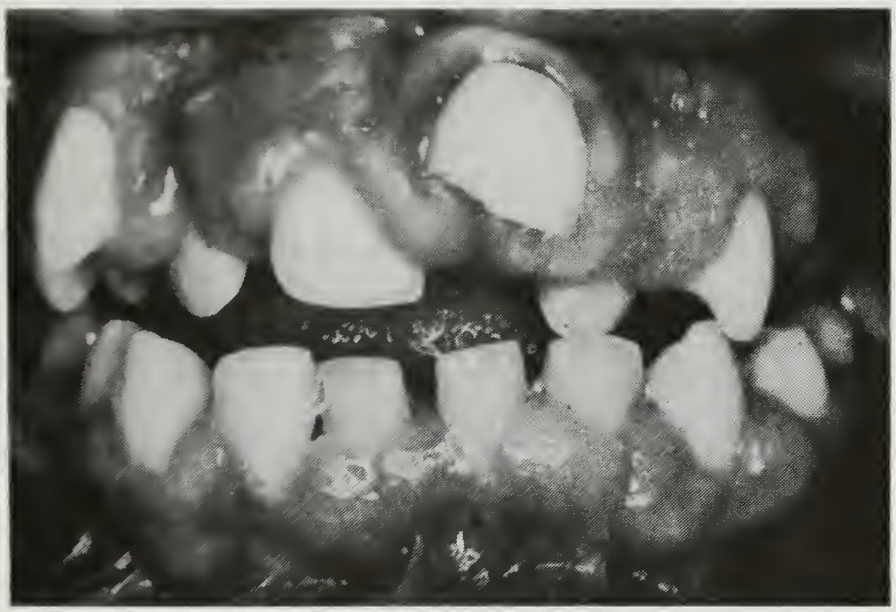


Figure 11: Lingual view of teeth and tissue prior to gingivectomy.

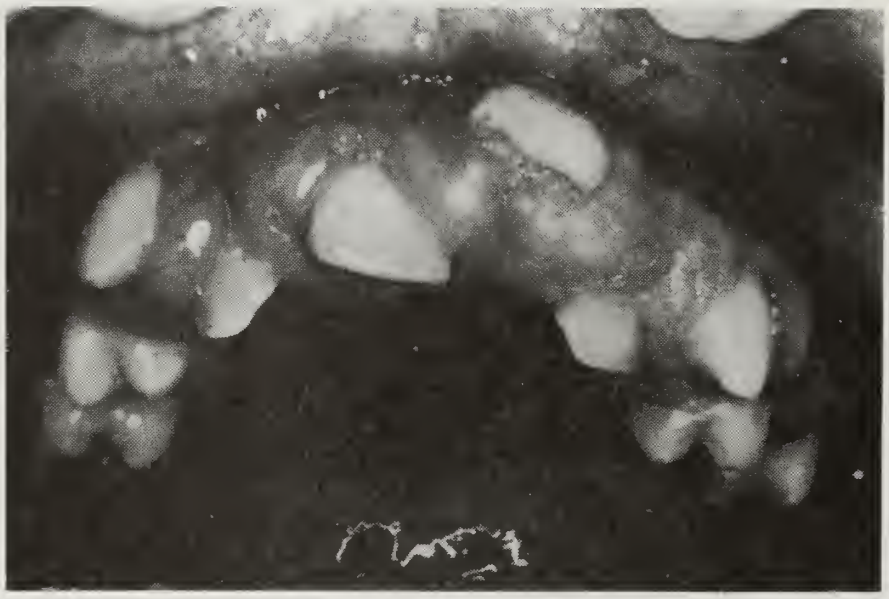


Figure 12: Stent prepared on stone model in two sections. Model prior to gingivectomy.

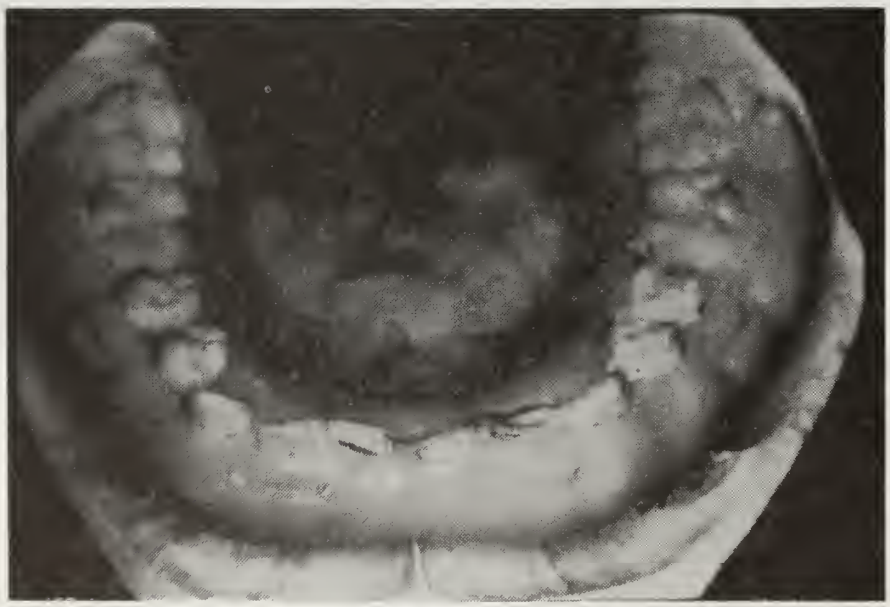


Figure 13: Stent holding periodontal packing around teeth following gingivectomy. Stent ligated with orthodontic wire.

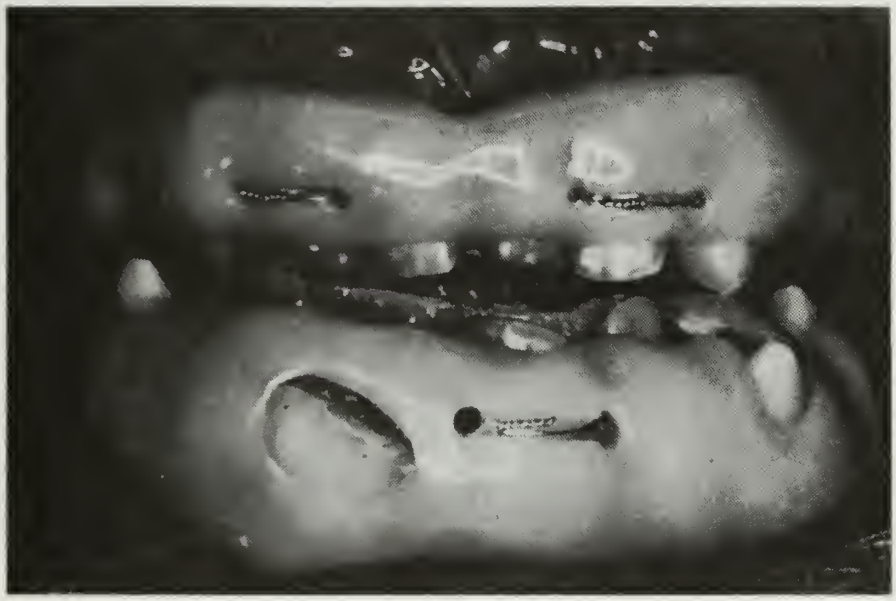
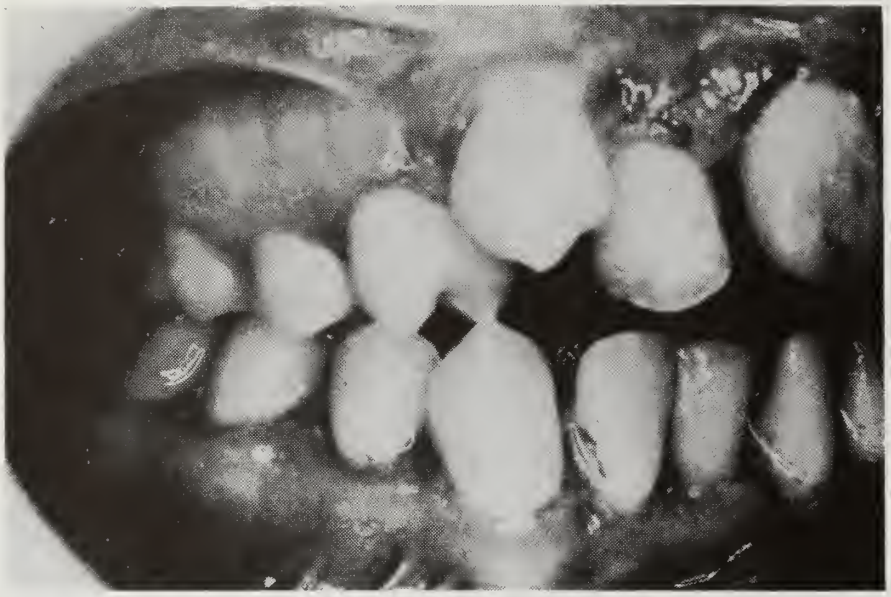


Figure 14a, b: Lateral views of gingiva one week post-operatively.



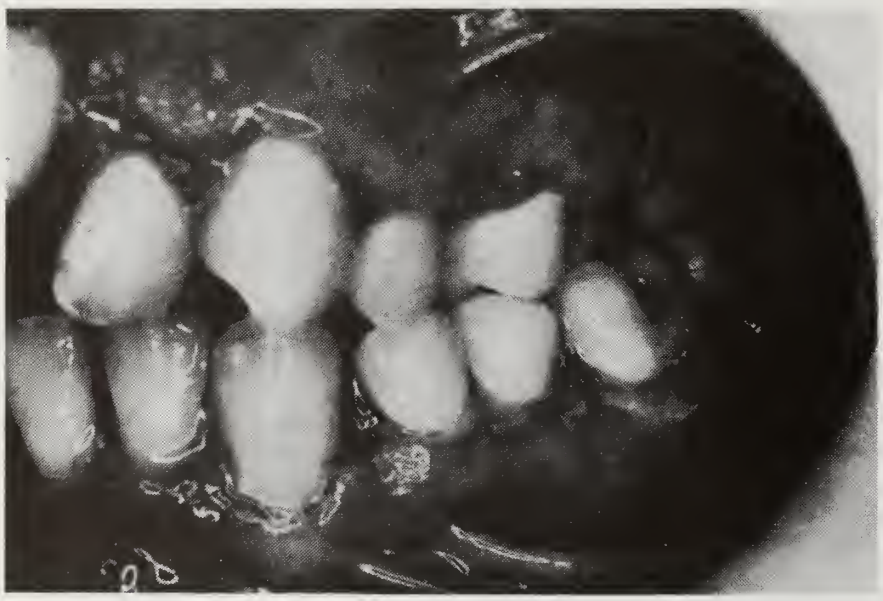


Figure 15: Anterior view of teeth and gingival tissue 3 weeks post-operatively.

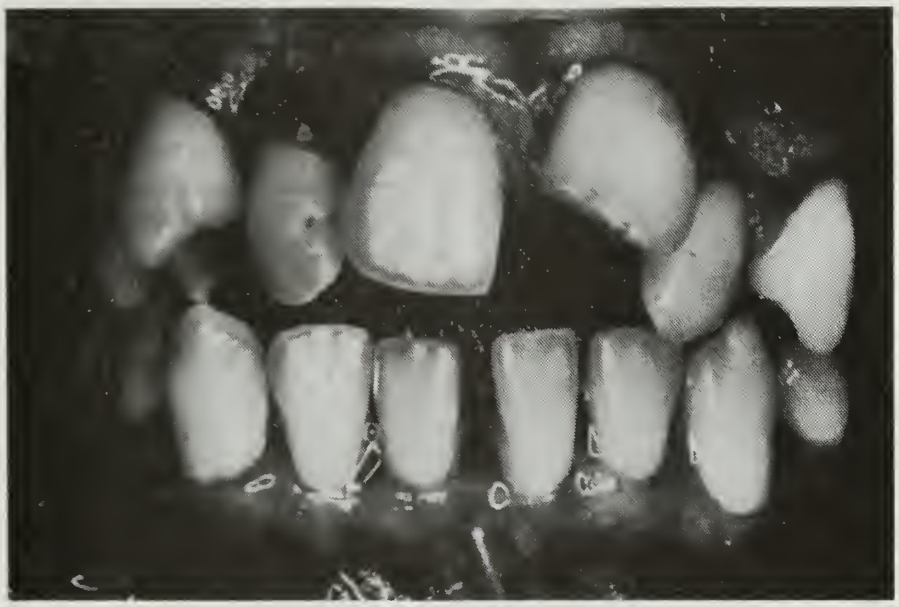


Figure 16: Three months post-operative. Note migration of centrals to a more normal relationship following removal of excess tissue.

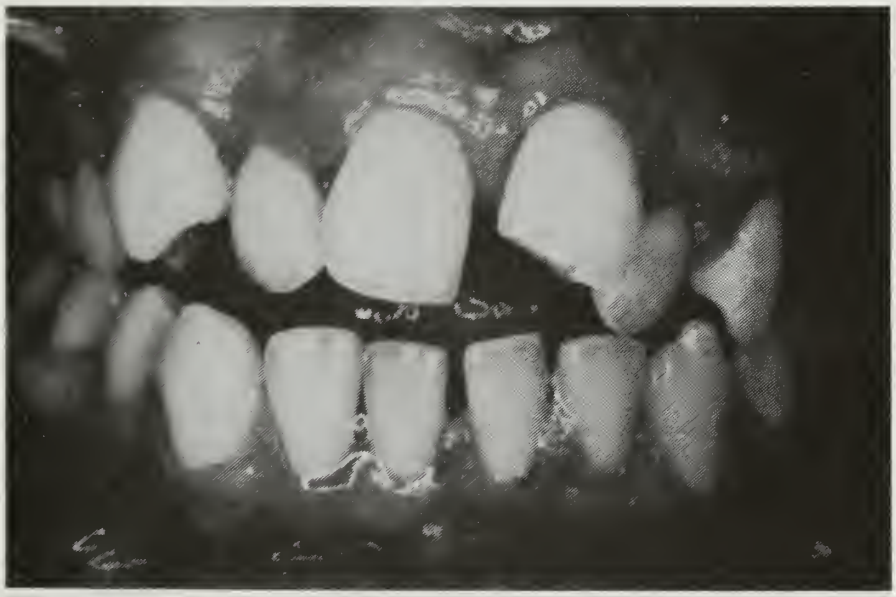


Figure 17a: 12 mm. distance between central incisors prior to treatment.

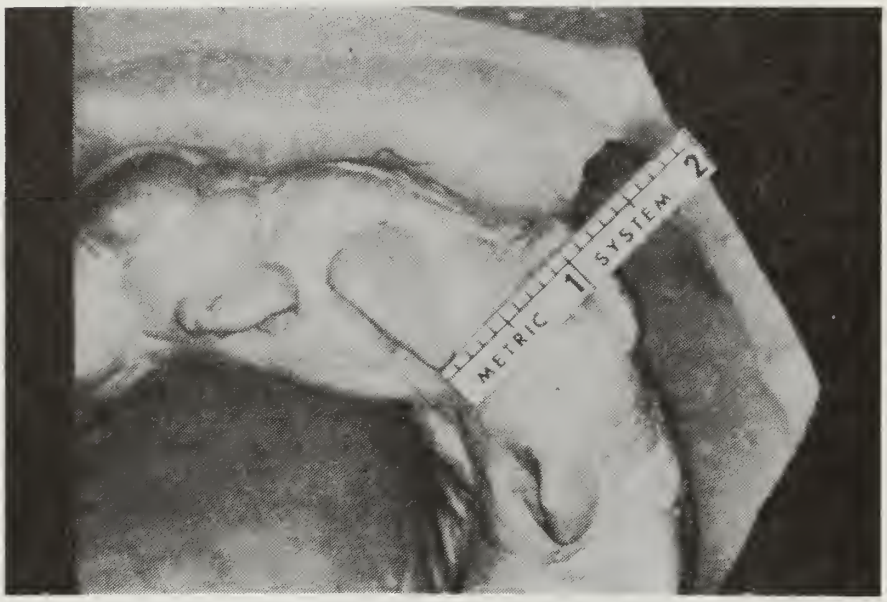


Figure 17b: Distance between central incisors has been reduced by 7 mm. following gingivectomy.

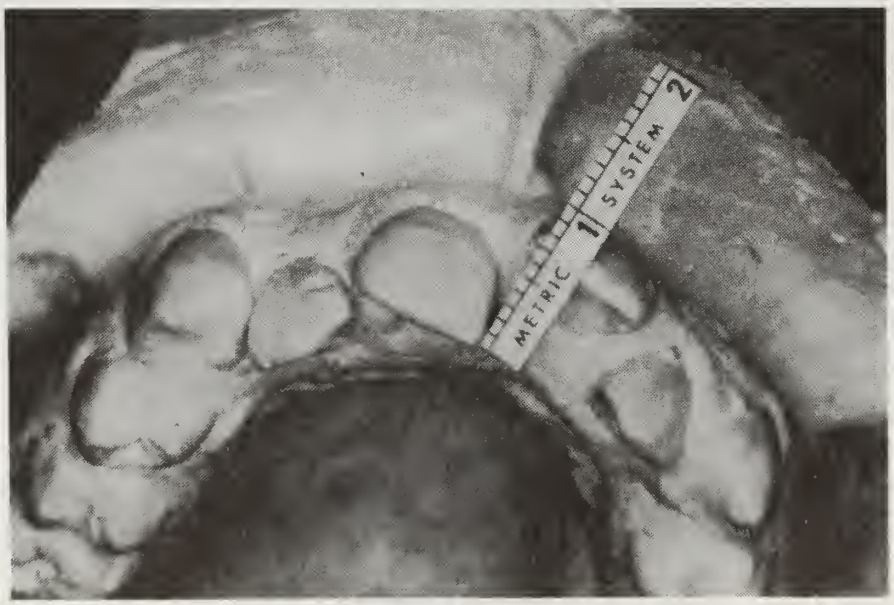
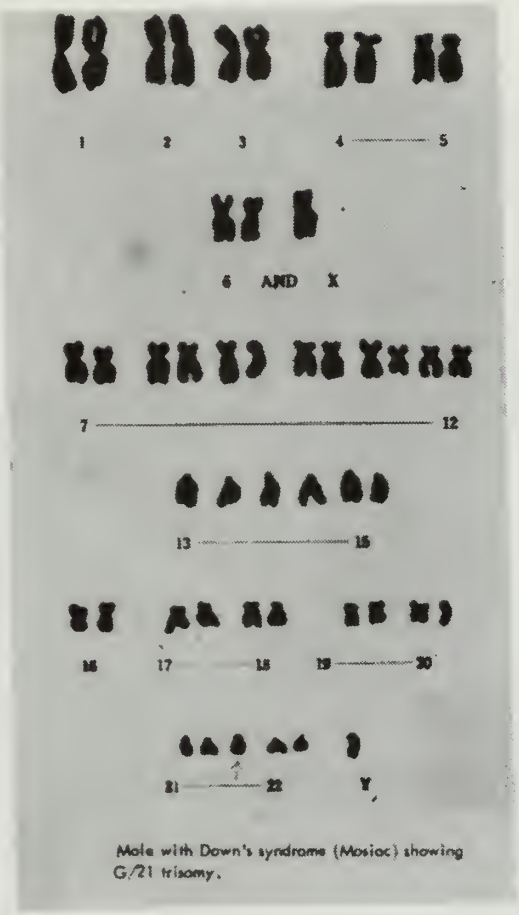


Figure 18: Karotype of male with Down's Syndrome. Note G/21 Trisomy.



Male with Down's syndrome (Mosiac) showing G/21 trisomy.

Figure 19: Ten year old patient with Down's Syndrome. Note midline defects in mandibular incisor region (2/3 bone loss) with superimposed chronic necrotizing gingivitis.

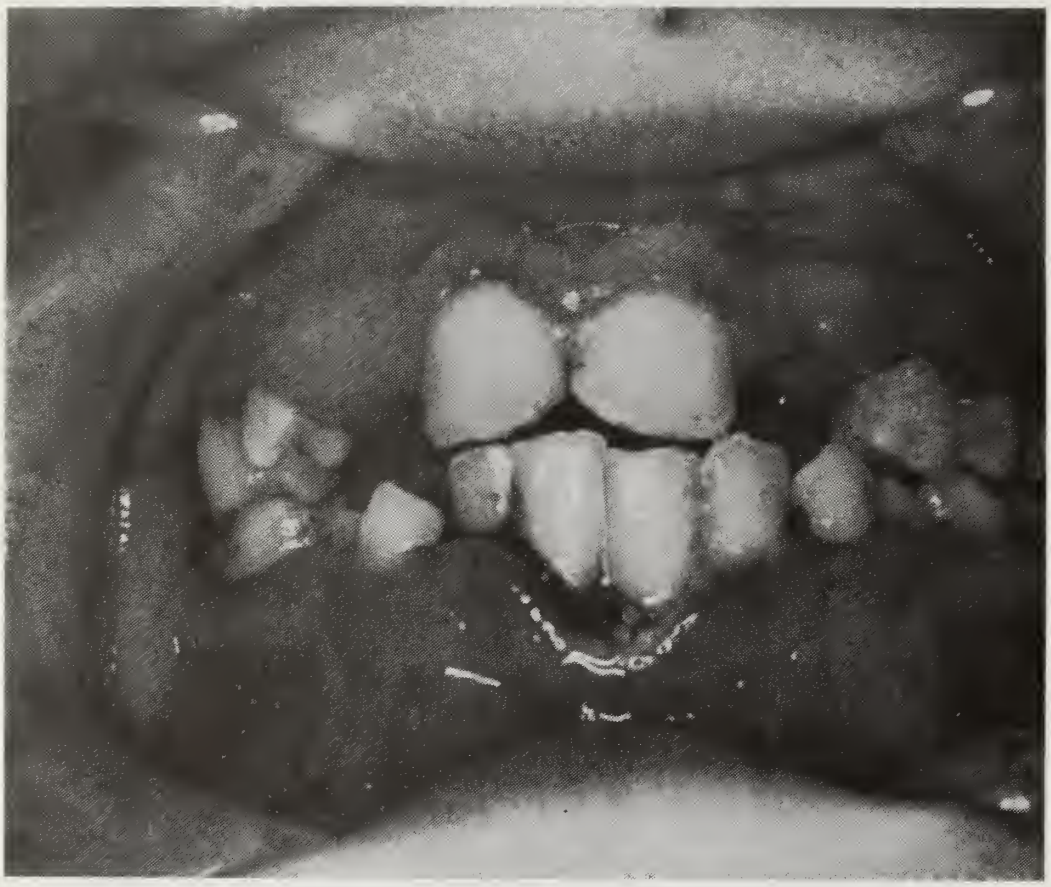


Figure 20: Generalized periodontal involvement in a five year old patient with Down's Syndrome.

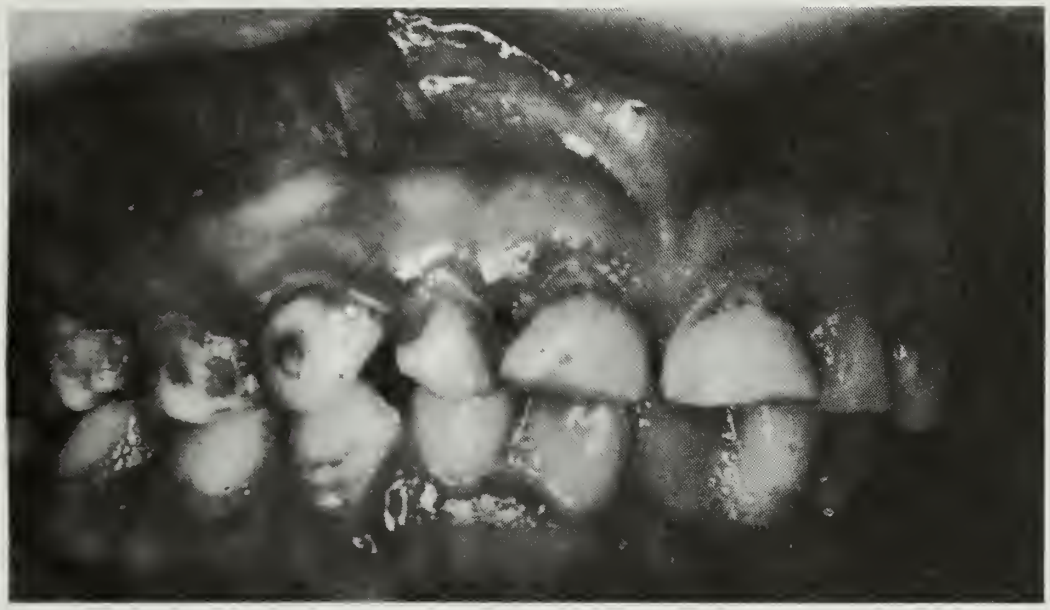


Figure 21: Proliferative gingival involvement on a four year old patient with Down's Syndrome. Note hypoplastic defects on incisal surfaces of maxillary central incisors.

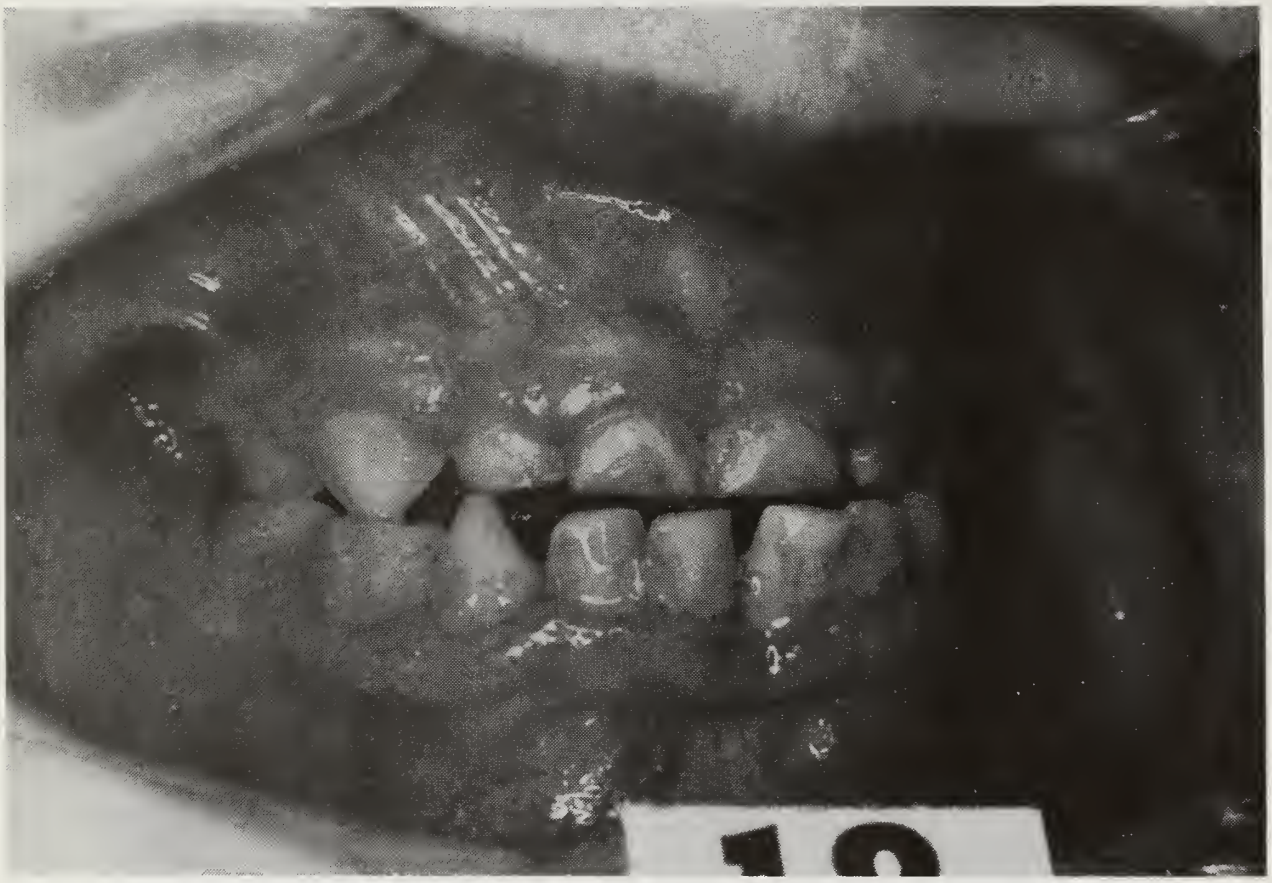


Figure 22: Generalized periodontal involvement in a young patient with Down's Syndrome. Note hypoplasia in maxillary central incisors.

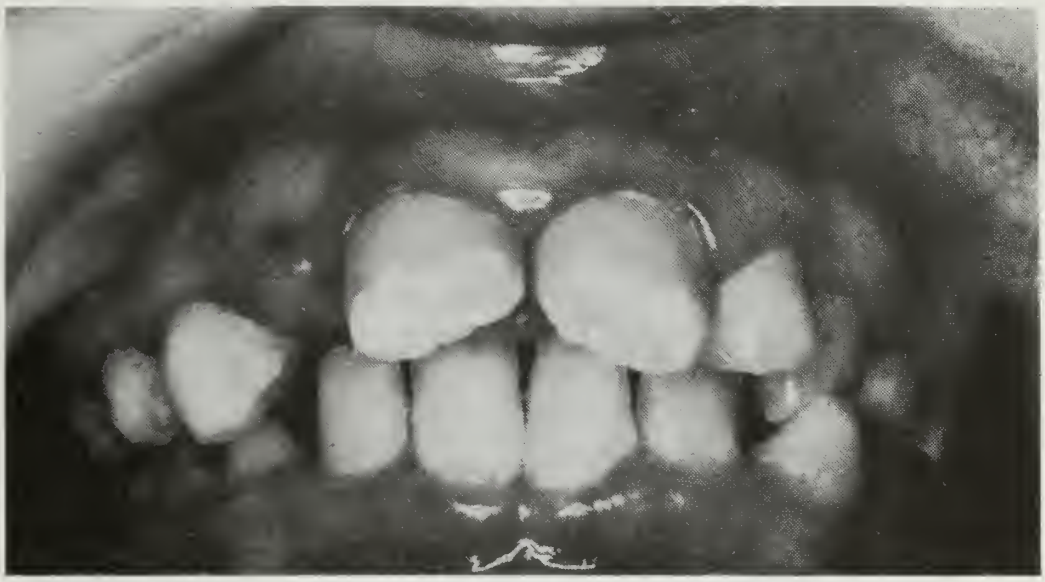


Figure 23: Full mouth roentgenographic survey in a seventeen year old individual, non-institutionalized, with Down's Syndrome. Note generalized bone loss throughout the mouth.

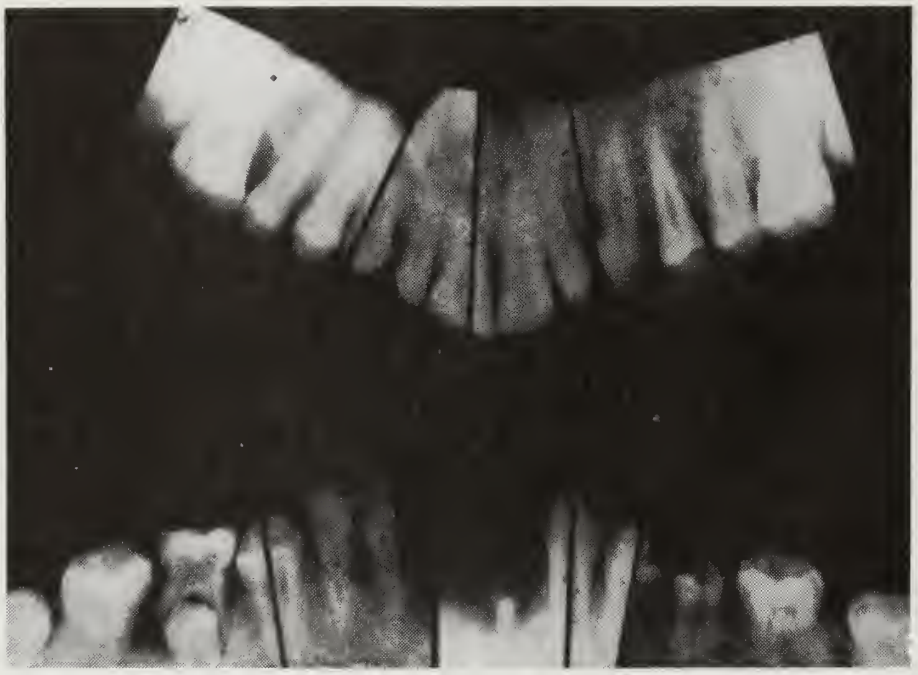


Figure 24: Young patient with Down's Syndrome. Note generalized periodontal involvement with chronic necrotizing gingivitis in mandibular incisor region. Also note anterior crossbite common in Down's Syndrome.

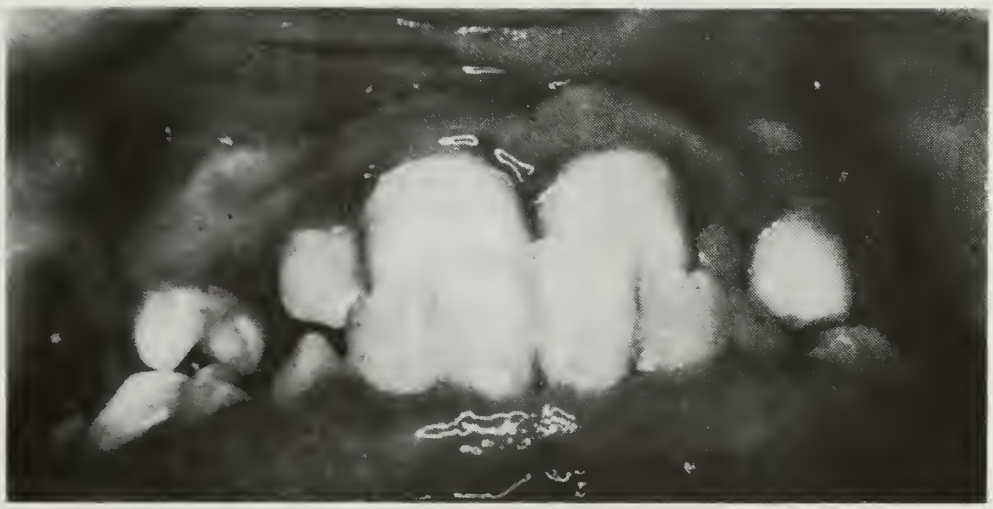


Figure 25: Radiographs of mandibular incisor regions of three male, non-institutionalized individuals, age 10-12 years, with Down's Syndrome. Note severe bone loss in mandibular incisor region.

Figure 26: Radiographs of mandibular incisor regions of three female individuals, age 9-12 years. Note severe bone loss in mandibular incisor regions.

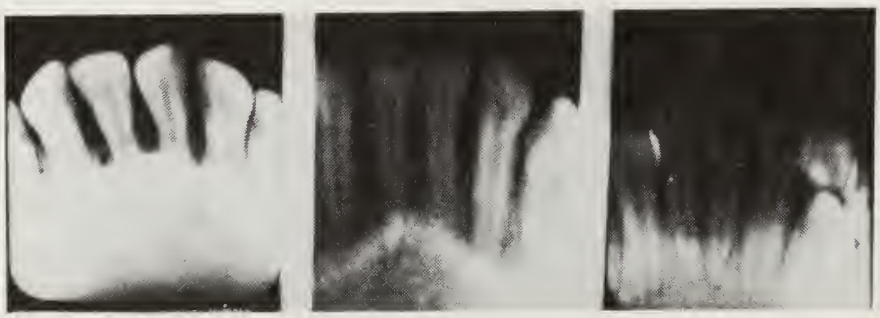


Figure 27: Fourteen year old non-institutionalized male with Down's Syndrome. Note bilateral crossbite and chronic destructive periodontal disease.

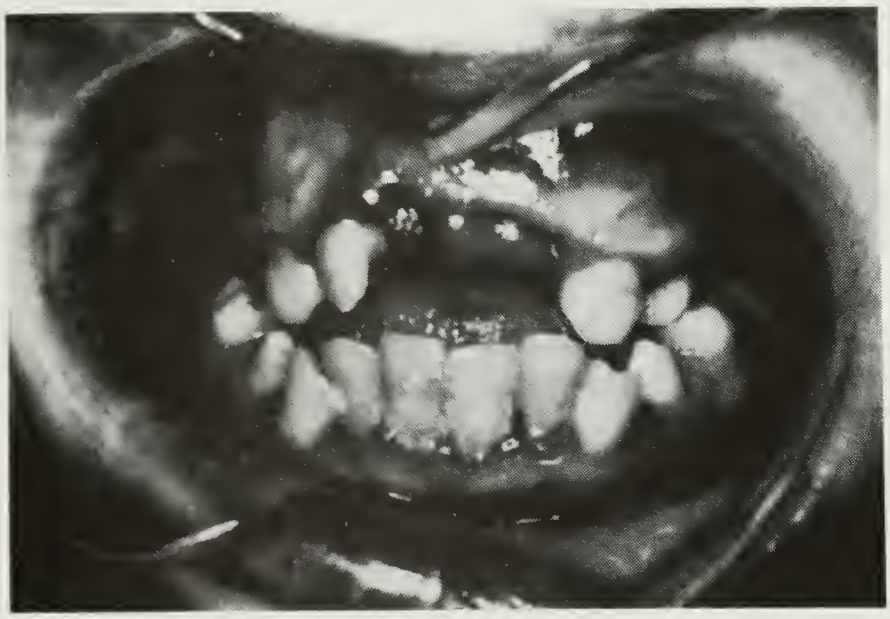
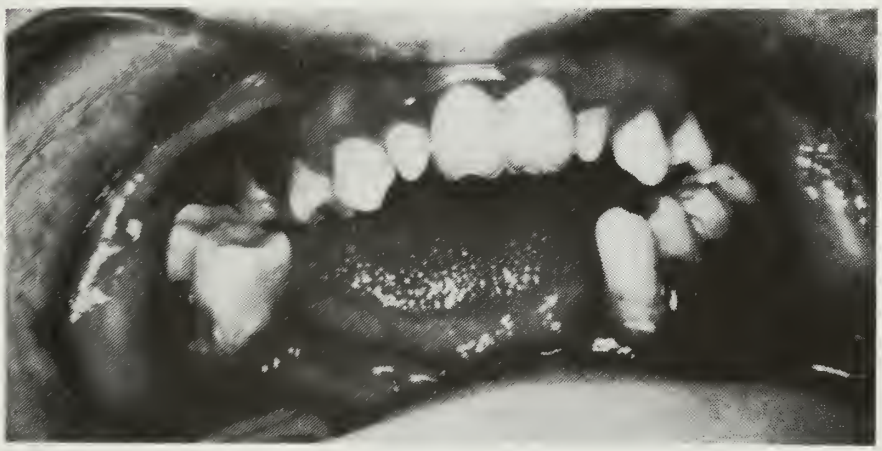


Figure 28: Nineteen year old, non-institutionalized male, with Down's Syndrome. Note severe occlusal disharmony with chronic destructive periodontal disease.



PREMEDICATION

Joseph P. O'Donnell, D.M.D., M.S.

Premedication can be of significant benefit in the behavior management of mentally retarded individuals who present for dental treatment. The goals of premedication are as follows:

1. To allay fear and anxiety

Example: Anxious patients tend to over-react to even the slightest painful stimuli. Such negative responses in a mentally retarded individual may preclude the performance of simple preventive or operative procedures on an ambulatory basis.

2. To reduce undesirable reflex activity

Example: Apprehensive patients tend to manifest an exaggerated gag reflex which can produce a major obstacle in accomplishing outpatient dentistry.

3. To reduce motor activity

Example: A patient may continue to bring his hands up into the operating field or twist and squirm despite instructions to remain still. Persons afflicted with tremors or spasticity may be unable to lie still for dental procedures without the aid of sedative agents.

4. To raise the pain threshold

Example: As mentioned above anxious patients have an altered perception of pain. In addition to producing calming effects, several premedicating agents can also raise the pain threshold to bring about a more receptive and treatable patient.

Routes of Administration:

Premedication agents can be administrated in several ways. The oral route of administration provides the easiest access and most convenience. For this reason it is the most popular choice when dentists wish to use pharmacologic agents to sedate mentally retarded patients.

Several factors must be considered, however, when selecting the oral route of administration. These factors include:

Palatability - Some handicapped patients may refuse or be unable to swallow tablets or capsules. Also, some drugs may not be available in liquid form and, if they are, the taste may not be acceptable.

Contents of stomach - If the patient's stomach is full, orally ingested pharmacologic agents may be poorly absorbed resulting in a diminished or delayed effect. For this reason, it is suggested that the patient refrain from eating for at least 4 hours prior to oral premedication.

Reduced effectiveness - Many drugs have a reduced effect when taken orally.

Parent or Direct-Care Staff Dependent - When oral premedication is prescribed, the dentist relies upon a third person, usually the parent or a direct care staff person, to administer the drug as ordered.

Prerequisites for Safe Administration

The dentists who elects to use premedication should follow several basic guidelines in order to maximize the safety of the patient:

Medical history - A detailed medical history is essential in order to rule out any contraindication for sedation. This history must include specific questions pertaining to drug allergies or sensitivities. If the patient is taking other medications, drug interactions must be considered. For example, tricyclic anti-depressants, phenothiazines and barbiturate agents are commonly utilized in the psycho-therapeutic management of mentally retarded individuals. The inter-relationships of these medications and the premedicating agents must be considered. If there are any questions pertaining to the patient's medical history or current medications, the patient's physician should be consulted.

Physical examination - The patient who is considered a candidate for premedication should have a physical examination with a physician within the past 6 months. This is important to rule out any underlying cardiovascular, respiratory or other medical conditions which could contra-indicate sedative agents. If the patient has a history of hepatitis, it is advisable to review liver function studies or a blood chemistry profile to insure that the patient's liver is functioning

properly. The importance of this point lies in the fact that many premedicating agents are detoxified and destroyed in the liver.

Read manufacturer's directions - When a particular pre-medicating agent has been selected for use, it is essential that the dentist review the pharmacology of that agent in detail. One of the best sources for the information is the product information insert that accompanies the drug or a current copy of the Physician's Desk Reference. The indications, contra-indications, precautions, adverse reactions and dosage ranges must be carefully reviewed.

Specific Antidote - If a narcotic analgesic is selected for premedication, the specific antidote or narcotic antagonist must be on hand in case of an adverse reaction. The narcotic antagonist of choice if naloxone hydrochloride (Narcan) (R)

Oxygen - Although a supply of emergency oxygen is essential in any dental office, it is particularly important if sedative agents are utilized, since many of these act as mild respiratory depressants.

Suction - Adequate high volume suction is also essential to ensure the rapid removal of saliva, debris or vomitus. Failure to do so could lead to airway obstruction, and respiratory complications.

Agents used for Premedication

The drugs most commonly used for premedication of handicapped individuals in the dental environment are generally classified into four major groups:

1. Sedative hypnotics

2. Psychosedatives

3. Antihistamines

4. Narcotic Analgesics

I. Sedative Hypnotics:

The drugs in this category are used to relieve anxiety through central nervous system depression. Their ability to produce sleep is dosage dependent, with larger doses resulting in profound sedation or hypnosis. Clinically, the main difference between this group and the narcotic analgesics is that they lack the ability to obtund the sense of pain without producing a definite impairment of consciousness.

A. Secobarbital (Seconal) (R)

Nembutal (Pentobarbital) (R)

Secobarbital and pentobarbital are short-acting barbiturates which are considered to be strong sedatives and hypnotics. Like all barbiturates they have no analgesic action and actually decrease the pain threshold. They should never, therefore, be prescribed as premedicating agents for any patient in pain.

Some patients receiving barbiturates may exhibit a phenomenon known as "paradoxical excitement" in which they appear stimulated rather than sedated. This response indicates that these agents are somewhat unpredictable for use as premedicating mentally impaired individuals.

Available Forms - Secobarbital

a) Capsules: 50 mg, 100 mg

b) Elixir : 22 mg/5cc

c) Suppository: 200 mg

d) Injectable : 50 mg/cc

Pentobarbital

a) Capsules: 30 mg, 50 mg, 100 mg

b) Injectable: 50 mg/cc

Recommended dosage

Children 12 and under: .5 mg/pound

Adult: 1 mg/pound

B. Chloral Hydrate - (Noctec) (R)

Chloral Hydrate is a safe and effective sedative hypnotic which has been used in medical practice for over 100 years. It quiets sensory and motor excitement to induce a calming effect. In therapeutic doses, respiratory and cardiovascular depression are not noted. Chloral hydrate is absorbed rapidly and is usually effective within thirty to forty minutes. It is, however, a gastric irritant and may produce nausea. The duration of action is four to eight hours.

Available Forms - a) Capsules: 250 mg, 500 mg

b) Elixir: 500 mg/5cc

c) Suppository: 650 mg

Recommended dosage: 10-20 mg/pound

II. Psychosedatives

The psychosedatives are comprised of a group of drugs that act as ataractic or tranquilizing agents to induce a calming effect. They are not cortical depressants, but rather suppress activity in sub-cortical areas of the central nervous system (limbic system) to bring about relaxation.

A. Hydroxyzine Pamoate and HCL (Vistaril (R) and Atarax)(R)

These drugs are quite effective in relieving apprehension while having little effect on the cardiovascular and respiratory system. They are good anti-emetic agents which makes them useful for a combination with nausea producing drugs such as Chloral Hydrate. They also produce a mild xerostomia (which is often quite desirable in treating the mentally impaired) and tend to potentiate barbituates and narcotic analgesics.

Available forms:	Vistaril:	a) Capsules	: 25, 50, 100 mg
		b) Suspension	: 25 mg/5cc
	Atarax:	a) Tablets	: 10, 25, 50, 100 mg
		b) Syrup	: 10 mg/5cc
Recommended dosage:		1 mg/pound	

B. Diazepam (Valium)

Diazepam is a member of a group of compounds called benzodiazepines with a broad spectrum of pharmacologic activity. Diazepam has many well known properties including anti-anxiety and sedative effects, anti-convulsive actions and muscle relaxation.

Available forms: a) Tablets : 2, 5, 10 mg

b) Injectable

Recommended dosage: a) Underage 12 : 2-5 mg p.o.

b) Overage 12 : 5-10 mg p.o.

III. Antihistamines

A. Promethezine Hydrochloride (Phenergan)

Although the antihistamines are seldom used alone for relaxation of anxious patients, they have many applications for use with other sedative agents. For example, Promethezine is an excellent anti-emetic and has been shown to potentiate the effect of sedative-hypnotics and narcotic analgesics.

One of its uses with the mentally impaired individual is to help reduce an exaggerated gag reflex.

Available forms: a) Tablets: 25 mg, 12.5 mg

b) Elixir : 25 mg/5cc

Recommended dosage: .5 mg/pound

IV. Narcotic Analgesics

Although the principal action of the narcotics is to relieve pain, they are also mildly sedative in action. They are central nervous system depressants and can cause severe respiratory depression.

A. Meperidine HCL (Demerol)

Meperidine is a potent narcotic analgesic with side effects of mild sedation. It is absorbed rapidly and has a duration of action of approximately 2 to 4 hours. Since Meperidine may produce nausea,

combining an anti-emetic agent with it may prove to be useful.

Available forms: a) Tablets : 50, 100 mg.

b) Elixir : 50 mg/5cc

c) Injectable

Recommended Dosage: .5 = 1.0 mg/pound

GUIDELINES FOR PREMEDICATION

1. Apprehensive Patients

A) Valium 5 to 10 mg p.o.

OR

B) Vistaril .5 - 1.0 mg per pound

2. Mildly Retarded and Mildly Resistive Patients

A) Chloral Hydrate 10 - 15 mg per pound p.o.

(Hypnotic dose 20-25 mg per pound)

AND

Vistaril .5 - 1.0 mg per pound p.o.

OR

Phenergan .5 - 1.0 mg per pound p.o.

3. Moderate to Severe Retardation and Resistive Behavior

A) Demerol .5 - 1.0 mg per pound p.o.

OR IM

AND

Phenergan .5 - 1.0 mg per pound p.o. or 1M

INTRAVENOUS AND NITROUS OXIDE/OXYGEN CONSCIOUS SEDATION

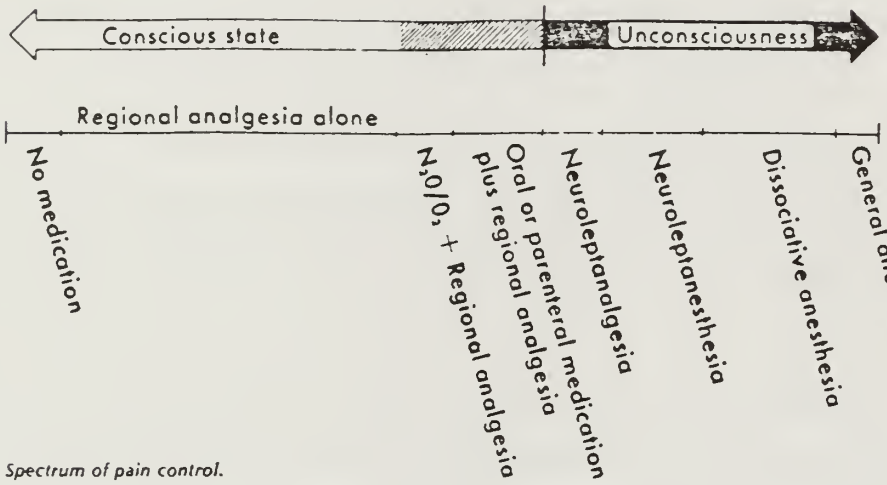
Michael P. Monopoli, D.M.D.

The dental practitioner who assumes the responsibility of treating the developmentally disabled individual has a dual role. Technically sound care as well as behavioral management must be provided. Physical handicaps, apprehension, and lack of understanding create needs not often seen in the general public. Quality dental treatment of the developmentally disabled patient is impossible unless those needs are met. A large segment of the special needs population cannot or will not accept routine dental care. Often conscious sedative medications can be a useful adjunctive management technique and allow a previously uncooperative individual to accept dental treatment comfortably.

Sedative medications can be safely and economically provided by a trained practitioner through inhalation or parenteral routes (or oral as previously discussed) in the dental office. Safety is maintained during procedures, since the patient remains conscious throughout treatment with protective reflexes intact. Dentists treating special needs individuals should have some form of conscious sedative technique in his/her armamentarium.

Often there is confusion concerning the indications for and use of conscious sedatives. The advisability of utilizing those medications in the dental office has been questioned. A few terms will be reviewed in an attempt to clarify that confusion. The terms are:

Conscious patient: one with intact protective reflexes, including the ability to maintain an airway, and who is capable of "rational" response to question or command.



Spectrum of pain control.

Analgesia: - The diminution or elimination of pain in the conscious patient.

Sedation: - The calming of a nervous, apprehensive individual by use of systemic drugs without inducing loss of consciousness. The agents may be given orally, patenterally, or by inhalation.

Hypnosis: - Condition of sleep produced by hypnotic drugs.

General Anesthesia: The elimination of all sensations, accompanied by a state of unconsciousness.

Coma: - A state of unconsciousness from which a patient cannot be aroused, even by powerful stimulation. [2, 5]

The significant factor differentiating dental sedative techniques from more risky general anesthesia is consciousness. The objective of conscious sedation is to administer the least amount of medication that will produce a relaxed and indifferent patient without producing unconsciousness. Conscious sedation occupies the grey area of a continuum, between extremes of full consciousness and total lack of consciousness, as illustrated above. The patient's status on that continuum is dose related. The patient must be carefully monitored to maintain consciousness at all times.

The rapid uptake and almost immediate symptom onset of nitrous oxide/oxygen and intravenous medications allow them to be used as effective conscious sedative agents. Unlike oral and intramuscular routes, inhalation and intravenous medications can be slowly administered in incremental doses, or titrated. The point at which a patient begins to feel symptoms and exhibits signs of sedation, or baseline, can be precisely reached.

As defined by Jorgensen,[7] first symptoms of light sedation appear (i,e. slight drowsiness, dizziness, or blurring vision). Thereafter, an additional measured amount of drug(s) is given to realize the full effect of light sedation. Baseline and titration are extremely important concepts in the production of a safe conscious sedation state.[5]

Nitrous Oxide/Oxygen

Nitrous Oxide/Oxygen (N_2O/O_2) is readily available in many dental offices. When mixed with oxygen, nitrous oxide can be inhaled through a nose piece. N_2O/O_2 is contraindicated in cases of pregnancy, severe COPD,* psychosis and nasal blockage. The complications of N_2O/O_2 use are few and easily avoidable with proper technique. Rapid diffusion of N_2O into the bloodstream induces baseline symptoms within minutes. N_2O/O_2 can be titrated, therapy avoiding nausea and excitation. The rapid diffusion of N_2O out of alveoli also concentrates the remaining O_2, creating a margin of safety against hypoxia, [3]. The patient should breath 100% O_2 at the termination of treatment to guard against the transient hypoxia which may result as N_2O diffuses rapidly back into the alveoli from the bloodstream[3].

N_2O/O_2 sedation requires behavorial management and profound local analgesia. The nose piece may be as upsetting to the parent as the dental treatment to be provided. If care is taken to gently acquaint the patient with the nosepiece he/she may accept its use. The pleasant smell of spirit of peppermint on the nosepiece and oral premedication may be helpful in some instances.[9]

If the nosepiece is accepted the patient should breath 100% O_2 for a short time. The patient respiratory tidal volume should be

*Chronic obstructive pulmonary disease

approximated by observing the reservoir bag. O_2 flow is then decreased while N_2O flow is increased (1 liter/min. increments) at approximately two minute intervals. Titration should cease when symptoms of CNS depression are noted (i.e. baseline). N_2O/O_2 flow should remain at that level until treatment is terminated and 100% O_2 is administered.

With behavior management and profound local analgesia, a number of previously uncontrollable social needs patients can be treated in the dental office. If a level of 50% N_2O is reached, however, and a patient is still unwilling to accept treatment, the patient should be dismissed and evaluated for use of another sedative technique.[5] In many cases intravenous conscious sedation is the treatment of choice for those patients.

Intravenous Conscious Sedation:

A trained practitioner can administer intravenous conscious sedative medications to the special needs patients in the dental office. Training may be obtained in a number of ways and does not necessarily require training in general anesthesia.[4,5] Special supplies required are minimal. A dental office should be equipped with a source of positive pressure oxygen, blood pressure cuff, airways and emergency medications regardless of intravenous medication use. A cardiac monitor is recommended but not mandatory. Although an operator/sedationist can provide comprehensive dental care to the general public, it is prudent to have an operator and sedationist available when treating special

needs patients. Unpredictable behavior can be controlled more easily and both patient and operator will benefit.

The intravenous conscious sedative technique most frequently employed are:

1) Valium
2) Valium and N_2O/O_2 [9]
3) Valium and Narcotic (+ scopolamine)
4) Valium and Methohexitol (atropine) [6,8]
5) Jorgensen technique (Nembutal, demerol, scopolamine)[4]

Medications are slowly titrated in all of the above techniques. When baseline is reached, an additional measured amount of medication is administered to fully develop the sedative effect. Vital signs and the patients level of consciousness are carefully monitored throughout the entire procedure. With profound local analgesia these techniques usually result in a patient who will accept dental treatment.

Problems encountered during the administration of intravenous medications most often result from lack of monitoring, overdose, or untreated hypoxia.[5] A sedated uncooperative patient generally requires more profound local analgesia, behavioral management, or a postponement of treatment, not more medication. Even with an uncooperative patient, however, intravenous conscious sedation "can make the impossible difficult"[10] and allow at least a through exam to access the need for general anesthesia or recall.

Conclusion:

Conscious sedative techniques can be an extremely useful adjunct to the dental treatment of special needs individuals. In the hands of a trained practitioner both inhalation and intravenous techniques can be safely and economically provided in the dental office. Careful adherence to the concepts of baseline, titration, and monitoring results in an atmosphere of comfort for both the patient and practitioner. Nothing, in dentistry, is more satisfying than treating a happy cooperative patient on recall, who no longer fears the dental office because of comfortable treatment, while sedated, he/she has had in the past.

Suggested Readings

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10. Wieman, H. Personal Communication 1981.

HOSPITAL MANAGEMENT

Robert R. Nersasian, D.M.D.

In order to provide comprehensive dental care of high quality, the dentist requires not only skill but also the unyielding cooperation of his patients. In the management of the physical and mentally handicapped patient this is the one factor that many times cannot be provided. To compensate, various forms of sedation have been devised including oral and intramuscular medications, inhalation analgesia, and intravenous sedation. Each of these techniques requires respectively higher degrees of training and represents higher degrees of risk. The physical status of the patient must also be considered. If one uses the analogy of cardiac patients and increasing degrees of oral surgical procedures, a similar format may be devised for the handicapped. In cardiac classification by symptoms, four groups exist: Class I - asymptomatic (single extraction-minor behavior disorder): Class II - symptoms while running (multiple extractions-seizure disorder, mild CHD): Class III - symptoms while walking (immediate denture - storage disorder, advanced CHD, seizure disorder): Class IV - symptoms at rest - (protracted odontectomies - cyanotic heart disease, chronic obstuctive lung disease, uncontrolled seizure disorders).

In those situations where office management becomes impractical and potentially injurious to the patient, hospital admissions and general anesthesia is mandatory. It is the purpose of this paper to present to the dentist the methodology for hospital staff privileges and the understanding with which to operate in this environment.

Application to a hospital medical staff is open to all duly licensed health professionals. Recent legislation by the Joint Commission on Accreditation of Hospitals has granted this open door policy. However, final acceptance is enacted at the local hospital level and is therefore not a guarantee. Most hospitals have a department of oral surgery or dentistry. Any licensed dentist may apply for staff privileges and will be judged on his/her credentials. Preference is given to those applicants who have either completed a dental internship or who have received a degree or certificate in a recognized dental specialty. The candidate is reviewed by a credentials committee comprised of hospital medical staff members. If accepted, a delineation of privileges is determined by the chief of the dental or oral surgery service based on the applicant's training and experience. Several categories of medical staff membership exist and include courtesy (nonvoting) and active (voting) staff appointments. If the dentist plans only occasional use of the hospital, the courtesy staff is the best choice. However, if the dentist's practice includes multiply handicapped patients requiring hospitalization, an active staff role is preferable.

Todays modern hospitals provide for varied admissions. The patient may be admitted the day before the procedure ("Overnight admission") to provide time for adequate work up and consultations. This admission would include patient types who may pose difficult medical control problems e.g. the diabetic, coagulopathies, or the advanced cardiopathy patient. "AM admission" i.e. arriving for admission the scheduled morning of surgery and staying overnight would include those patients

needing prolonged monitoring, due either to the procedure performed or the inability of the patient or his guardians to adequately care for him during the first 24 hour recovery period. "Surgicenter" or "Day Care" admission involves those patients who are medically stable and require only minor procedures which may include perio dental surgery, restorative dentistry or minor oral surgery. In this category the patient as in "AM admissions" enters the hospital NPO (nothing by mouth 8 hours) the morning of the scheduled procedure but, unlike admission, is discharged several hours after the operation. The patient is kept NPO to prevent aspiration during induction of anesthesia. In determining the form of admission the dentist must consider the procedure to be performed and the medical stability of his patient. In the latter instance, the American Society of Anesthesia Physician Status Classification may be beneficial. Most surgicenters permit only Class I and occasional Class II patients to be treated in this context. The ASA Classification is as follows:

Class I No organic, physiologic, biochemical or psychiatric disturbance

Class II Mild to moderate systemic disturbance

Class III Rather severe systemic disturbance or pathology from whatever cause, even though it may not be possible to define the degree of disability with finality.

Class IV Severe systemic disorder, already life-threatening

Class V Moribund patient who has little chance of survival.

E (Emergency Operation) Any patient in one of the classes listed above who is operated upon as an emergency is considered to be in somewhat poorer physical condition.

To admit patients to a hospital requires not only dental and medical skills, but an appreciation for the paper jungle. JCAH regulations and malpractice considerations demand acute detail to this paper bureaucracy. To admit a patient is a matter of a simple phone call, but like a trip cross country, a map or order sheet is essential to ensure the disposition and work up of the patient. In general, the admission order sheet should focus attention on the following: diet, bed and bath privileges, lab tests, consultations, and medications. In the simplest format, that of the surgicenter admission, a phone call achieves admission. In this instance, a physical exam is performed the morning of admission by the anesthesia department and a finger stick hematocrit and urine dipstick for sugar comprise the laboratory work-up. Remember, this admission is primarily for ASA Class I and occasional Class II patients. In the healthy patient, the physical exam and medical history rule out most problems. A negative urine surgar rules out insipid diabetes and normal hematocrit indicates oxygen carrying capacity sufficient to handle general anesthesia. If positive findings are seen in the physical exam or medical history, the above senario is modified in one of two ways: 1) last minute lab tests and consultations, 2) cancellation and rebooking under another admission type e.g. over-night. Diet in the day-care patient is not considered in the pre-op orders because the patient should arrive NPO and not eat until

after surgery; i.e. diet is considered in this context in the post-op orders. Bed and bath is based on common sense. If the patient is Class I, healthy and of normal mentality, he need not be restricted to bed. He may have full bathroom privileges. If handicapped, he may only be controlled by restricting to bed rest. Consultation with the anesthesiologist is required in all day care admissions. Due to the precise timing of these admissions, consultations with other specialists is usually impossible due to restraint of time.

The patient being admitted overnight or A.M. admission connotates either increased complexity of medical problems or increased difficulty of the proposed surgical procedure. In these cases, more advanced testing is indicated. Whenever possible, these test should be performed on an outpatient basis to save "hospital stay days." If possible, consultations should also be performed out-patient. However, there is latitude in the overnight admission for in-hospital consultation. In the A.M. admission, time does not permit for consultation.

Assuming more complex medical problems what tests should the dentist perform? This is difficult to answer because of the myriad of diseases and tests used for diagnosis. Whenever possible, it is advised to use the patient's private physician to perform the medical history and physical examination, as he is intimately familiar with the patient. The physician will advise and counsel on the necessary tests. Once again, if the patient can be seen before admission and the necessary documents completed, much time and effort can be saved. In a general sense, the following tests may be performed if the need is clinically and historically warranted:

1) CBC and differential

2) Chem profile

   a) 6/60 - BUN, BS, Electrolytes (Na,K,Cl, HCO_3) - if on antihypertensives or diabetic

   b) 12/60 - TOT Protein, albumin, calcium, phosphorous, uric acid, creatinine, total bilirubin, direct bilirubin, alkaline phosphatase, LDH, SGOT, Cholesterol, and triglycerides - if suspect liver disease or other systemic problems

3) Urine analysis - kidney status and UTI

4) Prothrobin time, Partial Thromboplastin time, and Platelets - if coagulopathy suspected

5) Chest X-ray - if evidence pulmonary disorder

6) EKG - Routine over age 40; Under 40, if suspect cardiac disease

7) Blood levels of such medications as dilantin and lithium to R/O toxicity and determine level of control

It is not in the context of this report to elucidate on all the above tests. Many excellent texts exist to indoctrinate the dentist in the use and interpretation of these laboratory exams.

In the overnight admission, attention must be given to diet. It should not differ from the patient's daily diet. If the individual is on a 1200 calorie low salt diet this logically follows through to hospital admission. In addition, any medications the patient takes as an outpatient should be considered while he is an inpatient e.g. Dilantin, phenobarbital, Mysoline or Depakene. Sedatives may be

necessary both for sleep and to relax the patient in this new environment. If prophylactic antibiotics are required, they are entered on the order sheet. A sample of overnight, surgicenter and A.M. admission orders are seen in Table #1.

Prophylactic antibiotics are an important consideration in this client population due to the high incidence of congenital heart disease. Any procedure that may involve bleeding and in which the patient has a heart murmur, should be premedicated. The AHA has developed two protocols: Regimen A and Regimen B. In most congenital heart disease, rheumatic or other acquired valvular heart disease, the drug of choice is penicillin and the method of delivery in the hospital setting is IM due to the NPO status. The dose is 600,000 units of procaine penicillin and one million units aqueous penicillin given 1/2 hour before surgery and followed post-operatively with 500 mg Pen V - QID x 8 dosages. In cases of penicillin allergy, erythromycin is the drug of choice.

Prior to surgery and any sedative premedication the patient, or in cases of legal incompetance, the patient's legal guardian must be informed of the proposed procedure, the results to be expected, a delineation and clear understanding of potential complications and an explanation of possible alternative treatments. This informed consent is mandated by the Supreme Court of Massachusetts and should not be only oral, but also written. Each hospital has its own consent form and a sample is seen in Table #2. A separate consent form is usually requested by the anesthesiologist in respect to his part of the

procedure. The importance of properly informed consent cannot be over-emphasized in this age of increased malpractice claims and exorbitant malpractice premiums.

Having admitted the patient, written the orders, evaluated the tests and consultations and received informed consent, the dentist, prior to surgery, should write an admission note. See Table #3. The admission note begins with the patient's name, age, and chief complaint: John Doe is an 11 year old male who presented with the chief complaint "my teeth hurt." History of the present illness (the chronology of the chief complaint) reveals the patient had been experiencing odontalgia from rampant decay over the past several months to the point where multiple dental abscesses have occurred necessitating hospital admission. A review of systems from head, eyes, ears, nose and throat to integumentary system is taken as part of the medical history. Significant findings are written: e.g. the patient is status post usual childhood diseases, denies allergies and is being treated with Ritalin for hyperactivity. A review of the physician's physical exam is listed and followed by a comprehensive dental history and exam. Pertinent laboratory and consultation reports are noted and a statement of the diagnosis and proposed treatment are made. A final statement concerning informed consent (i.e. given, understood, agreed and signed) ends the admission note.

A comprehensive oral examination begins with palpation for nodes and other visible or palpable lesions of the face, head and neck. The

examination then systemically proceeds from lips to tongue, floor of mouth, cheeks, hard and soft palates, tonsils and pharynx to end at the teeth and gingiva. See Table #3 - Admission Note.

The dentist is now ready to enter the operating suite. It is the doctor's ultimate responsibility to ensure that the necessary equipment is available. As your procedures increase in frequency, the operating room personnel will be able to have your equipment prepared before your arrival. The patient is now ready to be anesthetized. It is good policy to be in the room at this time to observe for any difficulties during induction. Once the endotracheal tube is placed and the patient connected to the anesthesia delivery system, you should scrub. A five to ten minute betadine or phisohex brush scrub from the finger tips to the elbows is required in all operating rooms. It is part of sterile technique. After the scrub, you will dry with a sterile towel, place on a sterile gown and gloves. Prior to the scrub, a mask and cap should have been in place. At this point, the patient's mouth may be prepped with a solution e.g. phisohex. The patient is then draped with sterile towels and sheets. The pharynx is then suctioned of all mucous and possible blood from the intubation and a gauze throat pack is placed to prevent posterior movement of foreign bodies. After all this preliminary work, the dentist is ready to begin the dental treatment.

Following surgery, an Operation Report must be dictated. It is a legal part of the hospital chart. The format is as follows and an example can be seen in TABLE #4:

Pre-OP Diagnosis	-	Dentigerous Cyst
Post-OP Diagnosis	-	Ameloblastoma (i.e. DX may change)
Procedure	-	Narrative Description
Surgeon	-	Dr. X
Assistant Surgeon	-	Dr. Y
Anesthesiologist	-	Dr. Z.
Anesthesia	-	Induction, type intubation, maintenance e.g. Pentothol/succinyl choline induction, nasoendo tracheal-intubation, maintenance with Halothane N_2O/O_2
Estimated Blood Loss	-	#cc - sponges and suction bottle minus irrigation
Fluids	-	#cc and type e.g. 700 cc P148D5W
Complications	-	Broken and unretrieved root tip
Adverse Reactions	-	Arrythmia secondary to Halothane inhalation

Sponge count - Correct i.e. pre-surgery count equals post-surgery count

Medications - Penicillin, 1 million units, Aqueous I.V.

Concise summarization of patients response and discharge to acute recovery area.

Example: Patient tolerated procedure well. Extubated in operating room and sent to recovery room with stable vital signs exchanging on his own.

Post operative orders direct the nursing staff in the treatment of your patient during his recovery. All pre-operative orders are cancelled by surgery and any orders you wish continued postoperatively must be rewritten. Typical dental post-op orders include:

1) Vital signs (V.S.) Q 15 min (every 15 minutes) till stable, then routine (every 4 or 8 hours)
2) Elevate head of bed 30° (semi Fowler position - decrease oral bleeding and throbbing)
3) ice to face (20 minutes on and 20 minutes off - if post surgery, this decreases pain and swelling)
4) Vaseline lips (minimize chapping)
5) Bite on gauze sponges 30-60 minutes PRN (as necessary) oozing -

6) Push PO fluids

7) Full liquid diet (soft or house diet depending on treatment)

8) Continue I.V. (e.g. Lactated Ringers) at #cc/hr (varies with patients weight, age and medical condition)

9) Meds:

   a) e.g. Demerol, 50 mg IM Q4H Prn Pain Severe

   b) Tylenol #3, one TAB PO Q4H Prn Pain Moderate

   c) Pen Vee K 500 mg PO Q6H

   d) Dilantin 100 mg PO TID

10) Discharge Order (if patient is a day care patient)

   a) Discharge to home when stable, Rx's Penicillin (500 mg x 40) and Tylenol 3(12) plus home care instructions and return appointments on front of chart.

If the patient is an overnight admission, a hypnotic may be prescribed on the order sheet to aid in sleep such as Dalmane 15 or 30 mg PO HS (hour of sleep). In addition, I.V. orders must be rewritten daily and narcotic orders every two to three days. If the I.V. is to

act only as a life-line and fluid overload is to be prevented, the I.V. order can be written:

Run Lactated Ringers to KVO (keep vein open) or place heparin lock (needle with 1000 u heparin to prevent clot) - maintains life-line without I.V. tubing.

Attention should be paid to either strict bed rest or ambulate ad lib which thereby includes full bathroom privileges. Diet may be advanced daily as the patient condition permits. In the multiple day admission daily rounds are recommended to adequately monitor the patient. Progress notes are to be entered at each patient visit and should include date, time seen, vital signs, patient complaints and statement of patient condition and plans for next day:

3/30/84 6:00 PM Patient alert, V.S. WNL (within normal limits). Afebrile. No complaints. No bleeding/oozing. Moderate edema left face. Good PO intake: Voided, Ambulated. Nauseous. Plan evaluation AM for discharge.

Sign your name

JCAH regulations require overnight admissions to have a discharge note. The format varies, but in general is as follows:
(may be dictated as the Op and Admission notes).

Date of Admission

Date of Discharge

Diagnosis

Associated Diagnosis

Chief Complaint

History of Present Illness

Medical History - Significant findings

Physical Exam - Significant findings

Dental Exam - Significant findings

Laboratory Tests - Significant findings

Operations

Adverse Reactions

Hospital Course - e.g. admitted 3/29/84, worked up routine tests, taken to O.R. 3/30/84 for restorative and oral surgery procedures and discharged uneventfully on 3/31/84 with Rx penicillin and Tylenol 3, prognosis excellent. Return office appointment 4/6/84, Home care and diet instructions given.

a) Chronology of admission

b) Discharge orders

c) Discharge drugs

d) Condition on discharge

e) Follow-up

The average healthy 70 kg male loses approximately 2000-2500 ml of water daily. This occurs as follows:

Urine	1200-1500 ml
Respiration	400 ml
Sweat	600 ml
Feces	100 ml

The daily Sodium loss is 40-90 meq and potassium loss is 20-60 meq Average blood volume is approximately 70 ml/kg body weight or approximately 5000 cc in the 70 kg male. About 60% of total body weight consists of water. Intracellular water is 45% by bodyweight and 20% is extracellular. Of this extracellular component 5% is intravenous and 15% is interstitial. During surgery, fluid loss is isotonic and due to dehydration, sweating, edema, and ventilation through the endotracheal tube. This exracellular loss is independent of and in addition to blood loss and, if not replaced, the interstial fluid department becomes depleted. The average loss of fluid is 5-10 ml/kg/hr. In addition blood volume decreases 5% after 8 hours of NPO. In the 70 kg male, 5000 ml x 5-10% = 250-500 ml. In summary, the minimal replacement for a 70 kg male 30 minute dental case is 500 ml (250-500 ml for NPO state plus 5-10 ml x 70 kg/hr [350-700 ml]) during surgery. Replacement is accomplished with a balanced electrolyte e.g. P148 D5W or D5WLR. If blood is lost it should be replaced in addition to NPO and operating fluid loss with a balanced solution at the rate of 1.5 to 2.0 cc per 1.0 cc blood loss. When blood loss is expected to exceed 1 unit (500 ml) the patient should be typed and crossmatched for replacement with whole blood or packed red blood cells. Postoperatively the 70 kg male's I.V. should be run at 100-125 cc/hr, if there are no cardiopulmonary problems. (2400-3000 cc which equates to average daily loss). Balanced electrolyte solutions contain adequate sodium replacement. Potassium takes several days to deplete and would not supplemented until the second to fourth postoperative day and then only if the patient is still taking nothing by mouth. It is important to cut down the I.V. flow as

the PO intake increases. In children, an average of 25cc per pound per 24 hours is an approximation for fluid replacement. (e.g.: 50 lbs x 25 cc = 1250 cc/day or 50 cc/hr). Urine output should be monitored for fluid overload and should be a minimum of 50 cc/hr or 1200-1500 ml/day. Wet sounding lungs, bladder distention, hyponatremia, and decreased urine specific gravity may be other signs of fluid overload.

Postoperatively, most patients require analgesic drugs. An analgesic medication obtunds the perception or interpretation of pain without producing unconsciousness. The pain threshold is raised. There are two main classes of analgesics non-narcotic and narcotic analgesics.

The historical favorite among the non-narcotic analgesics is the Salicylate - Aspirin. It has analgesic, antipyretic and anti-inflammatory properties and acts as an antiprostoglandin. Aniline derivatives e.g. acetominophen, share similar properties with the salicylates, but unlike salicylates, do not produce adverse effects in platelet interactions, especially in susceptible individuals. Newer antiprostoglandins e.g. Motrin (400 + 600 mg) and Dolobid (250-500 mg) combine the antiprostoglandin properties of salicylates with the analgesic effects of codeine. Propoxyphene compounds are also in this class and have various clinical usages.

Among the narcotic analgesics, morphine is the keystone. It is reserved for severe irretractable pain and is administered in dosages of 8-15 mg IM. Among the commonly used injectable narcotics, Demerol and codeine are at the top of the list. Demerol can be administered IM in dosages of 25, 50, 75 or 100 mg. Codeine is given S.C. in dosages of 1/2 or 1.0 gr and does not produce respiratory depression seen

occasionally with Demerol. Fentanyl has one hundred times the potency of morphine but does not have a role in postoperative analgesia. Nubain is a newer injectable narcotic with low abuse potential. It produces less respiratory depression than morphine but does produce sedation 36% of the time. It is given IM, IV, or S.C. at the dose of 10 mg/70 kg every 3-6 hours. The above medications are reserved for severe pain.

When postoperative pain is expected to be mild to moderate, oral narcotics may be used. Codeine may be given in its pure form from 1/8 to 1.0 gr. PO Q4H. Many compounds have been amalgamated with codeine to produce a myriad of drugs: Tylenol with codeine, Synalgos DC, Empirin Compound, Phenaphen with Codeine, Empracet, Fiorinal, Ascodeen 30 etc. Each has subtle differences in compounding and the clinician must decide which choice is preferred in each patient instance. Considered more potent than codeine PO is Demerol 25-50 mg tablets. It can be taken with ASA or Acetominophen to increase its potency. Patients allergic to codeine should not be allergic to Demerol. Other P.O. analgesics include Percodan, Percocet (acetaminophen), Tylox (acetaminophen) and Percodan-demi (1/2 strength oxycodone). These drugs are all semi-synthetic opium derivatives that produce analgesia and occasional sedation. They are frequently abused substances. One tablet every 4-6 hours should provide adequate pain relief from oral procedures. Dilaudid (2-4 mg PO Q4-6H) is a potent synthetic opiate derivative with ten times the potency of morphine and has no role in post-operative dental cases. Vicodin is a newer semi-synthetic narcotic that has

surpisingly not been classified as a class II narcotic. It is a hydrocodene Bitartrate Acetaminophen Compound taken 1 to 2 tabs Q4-6H. It may produce dependence and is additive other CNS depressants.

It is hoped that the reader of this chapter has gathered some insight to the methods and materials of the hospital setting. The chapter is not intended to totally educate but rather to enlighten. Sound judgment, good intuition, foresight and wisdom do not come about overnight by reading a book or adopting a method of discovering a formula. These invaluable assets on which our vital predictive processes in life are based, constitute a whole lifelong process of self-education. It is not easy, but it is the only way.

TABLE 1

USE NAME PLATE OR PRINT PATIENT ID HERE

Diagnosis:
A.M. Admission - Decayed teeth, prolapsed Mitral Valve

PHYSICIAN'S ORDERS

ASA

DRUG ALLERGIES

ANOTHER BRAND OF DRUG IDENTICAL IN FORM AND CONTENT MAY BE DISPENSED UNLESS CHECKED ☐

Date & Time Ordered	ORDERS AND SIGNATURE	Signature of Physician and Nurse attending to order
3/30/84	Admit AM Admission Dental Service	
	1) Keep NPO - Surgery this A.M.	
	2) CBC	
	3) Urine	
	4) Chm 6/60	
	5) EKG	
	6) Pre-Op Anesthesia Consult	
	7) Meds:	
	a) 600,000 units Procaine Penicillin and one million units Aqueous Penicillin IM on call to OR	
	Dr. X	

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PHYSICIAN'S ORDERS

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TABLE 1

SURGICENTER
Diagnosis:
Decayed teeth, Seizure Disorder

USE NAME PLATE OR PRINT PATIENT ID HERE

DOE, John
3792A

PHYSICIAN'S ORDERS

Codeine, ASA
DRUG ALLERGIES

ANOTHER BRAND OF DRUG IDENTICAL IN FORM AND CONTENT MAY BE DISPENSED UNLESS CHECKED ☐

Date & Time Ordered	ORDERS AND SIGNATURE	Signature of Physician and Nurse attending to order
3/30/84	Admit Surgicenter, DENTAL SERVICE	
	1) Finger Stick Hematocrit	
	2) Urine dip stick for sugar	
	3) Pre-Op Anesthesia evaluation	
	Dr. X	

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PHYSICIAN'S ORDERS

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TABLE 1

OVERNIGHT ADMISSION

Diagnosis:
Decayed Teeth, Congenital Heart Disease, Hypertension

USE NAME PLATE OR PRINT PATIENT ID HERE

PHYSICIAN'S ORDERS

No Known Allergies (NKA)

DRUG ALLERGIES

ANOTHER BRAND OF DRUG IDENTICAL IN FORM AND CONTENT MAY BE DISPENSED UNLESS CHECKED ☐

Date & Time Ordered	ORDERS AND SIGNATURE	Signature of Physician and Nurse attending to order
3/30/84	Admit Overnight DENTAL SERVICE	
	1) Strict Bedrest	
	2) House diet	
	3) CBC and differential	
	4) Urine Analysis	
	5) Chm Profile	
	6) EKG	
	7) Chest X-Ray	
	8) Consult Dr. Beat Cardiology	
	9) Consult Dr. Gas Anesthesia	
	10) NPO After Midnight for surgery 3/31/84	
	a) Dalmane 30 mg PO HS	
	Dr. X	

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PHYSICIAN'S ORDERS

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TABLE 2

CONSENT TO OPERATION OR OTHER PROCEDURE

Patient's Name	Age
Doe, John	4/10/52
Record No.	**Date**
3792A	

Patient Identification

Dr. Doe has explained that the above-named patient has the following condition(s) (Explain in Lay Terms)

Bad teeth

and that the proposed procedure(s) for treating/diagnosing the condition(s) is/are

Extraction teeth #1, 3, 7, 8, 9, 10, 11; Restorative dentistry cleaning

Explain in Lay Terms - State if Sterilization, Abortion and/or Amputation is Anticipated.)

I have also been informed of: (1) the potential benefits of the proposed procedure(s); (2) the alternatives; (3) the prospects of success; (4) the reasonably anticipated consequences if the procedure is not performed; and (5) the major risks involved in the proposed procedure. I have been given the opportunity to ask questions and have received satisfactory answers. I understand the necessity for the administration of anesthesia and blood transfusions, when deemed appropriate.

I am aware that in the practice of medicine, unexpected complications may occur. I acknowledge that no guarantees have been made to me concerning the results of the procedure(s).

I hereby authorize Dr. _____ and his delegated associates to perform and/or assist in the proposed procedure(s) described above.

If any condition is found at the time of the procedure that was not recognized before but which requires attention, I hereby authorize any procedure which in his professional judgement is necessary or desirable to remedy the condition.

I hereby authorize the taking of photographs or films during the procedure and their use for teaching and research purposes.

I hereby authorize the Hospital to dispose of any tissue necessarily removed as a part of the procedure(s) for diagnostic and research purposes.

I certify that I have read and fully understand the contents of this form, that the disclosures referred to above were made to me, and that all blanks and statements requiring insertion or completion were filled in before I signed my name below.

Signed: _____ WITNESS

Signed: _____ Patient or Parent/Guardian*

I certify that I have made the disclosures referred to above and have given the patient the opportunity to ask questions.

Signed: _____ Doctor

*Necessary if patient is under 18 years or incompetent.

NOTE: Consent of parent/guardian not sufficient for sterilization.

H1-200(7/81) - 151480

HOSPITAL TABLE 3

NAME UNIT NO.

DOE, JOHN ADMISSION NOTE: 2/13/84 A55344

CHIEF COMPLAINT: John is a 23 year old Caucasian male suffering from mental retardation. He was referred to us through his dentist Dr. Sunday for a chief complaint of pericoronal infections of the teeth 17 and 32.

PAST MEDICAL HISTORY: Reveals status post usual childhood diseases. He does have severe mental retardation and it is impossible to examine or treat him in a chair position. He is currently maintained on Dilantin and Valium under the care of Dr. Swaze from Newburytown.

PHYSICAL EXAMINATION: Reveals a well nourished, well developed 23 year old Caucasian retarded male. His weight is 118 pounds, height 63 1/2 inches. He has had a recent physical by both his physician and anesthesia at Hospital.

Heart presents with normal sinus rhythm without murmurs.

Chest clear to auscultation and percussion.

ORAL EXAMINATION: Reveals the lips to have a well defined vermilion border, no masses or lesions noted. Tongue well papillated. Full range of motion. Normal sensory and motor function. Cheeks and floor of the mouth are supple with clear salivary flow. Hard and soft palate, tonsils and pharynx are within normal limits. Gingiva indicates a chronic generalized gingivitis condition. There is also marked Dilantin hyperplasia. There are pericoronal infections associated with impacted teeth 32 and 17.

We are admitting John to Surgicenter Hospital. Under general anesthesia he will undergo examination of diagnosis, full mouth dental X rays and necessary treatment including odontectomies and cleanings.

The procedure has been explained to patient and parent, agreed and understood, permission slip has been signed.

RRN:NSMT:saf
D: 2/13/84
T: 2/15/84

WILSON WHO, D.M.D.

PERSONAL HISTORY and PHYSICAL EXAMINATION

TABLE 4

PATIENT'S NAME (Last,First)	MEDICAL REC. No.	OP DATE	SURGEON
DOE, JOHN	A55344	2/13/84	WILSON WHO, D.M.D.

FIRST ASSISTANT	ANESTHETIST	ANESTHESIA
DR. SUNDAY		Pentothal, Succinylcholine Induction, nasoendotracheal intubation, maintenance with Nitrous Oxide, oxygen.

Pre-operative Diagnosis Impacted wisdom teeth, #'s 1, 16, 17 and 32.

Chronic gingivitis and Dilantin hyperplasia.

Postoperative Diagnosis The same.
Name of Operation

The patient was prepared and draped in the customary manner. Prior to surgery a full mouth dental X-ray series was taken revealing 4 bony impacted third molars. There were no acute carious lesions noted. An oral dental exam was then performed indicating advanced gingival disease and Dilantin hyperplasia. At this time surgery commenced with a routine mucoperiosteal flap from left ascending ramus, anterior and buccal to first and second molar embrasure. Flap was reflected and held with a Selden. With round bur, Hall drill, and irrigation, buccal cortex was removed. Tooth was identified, split and removed in two pieces with straight and inclined plane elevators. All follicular tissues were enucleated and rough bone edges filed smooth. Area irrigated, flaps coapted and sutured with 3-0 Vicryl suture. At this time an incision was made over the left retrotuberosity of the maxilla, anterior and buccal to first and second molar embrasure, flap was reflected and held with a Selden. With monobevel chisel and mallet, buccal cortex was removed, tooth was identified and with Cogswell-B elevator tooth luxated from its crypt. All follicular tissue enucleated, rough bone edges filed smooth, area irrigated, flap coapted with sutures with 3-0 Vicryl suture. Sponges placed left mouth.

Surgery now commenced right mandible where again a routine mucoperiosteal flap was created from the ascending ramus anterior buccal first and second molar embrasure, flap was reflected and held with a Selden with round bur and Hall drill and irrigation-buccal cortex was removed. Tooth was found to be a lingual horizontal impaction, the crown was then split, removed in 3 pieces with straight and inclined plane elevators. Root tips were then split with fissure bur and removed in two pieces with a Cogswell-B elevator. All follicular tissues were enucleated. Rough bone edges filed smooth. Area irrigated. Flap coapted and sutured with 3-0 Vicryl suctures. Finally incision was made over the right retrotuberosity of the

maxilla, anterior and buccal to first and second molar embrasures. Flap was reflected and held with a Selden. With monobevel chisel and mallet, buccal cortex was removed. Tooth was identified and with straight and inclined plane elevators luxated from its crypt. All follicular tissues enucleated. Rough bone edges filed smooth, area irrigated, flaps coapted and sutured with 3-0 Vicryl suture. At this time, Dr. Sunday performed a full mouth examination and a full mouth hand scaling and curettage. At this point throat pack was removed, oral and nasopharynx was thoroughly suctioned. The patient tolerated the procedure well, extubated in operating room and sent to recovery room with stable vital signs, exchanging on his own.

CASE-FINDING AND REFERRAL

Katherine M. Pelullo, R.D.H., M.S.

The historical perspective has indicated that individuals who are developmentally disabled suffer from a lifetime of dental disease and neglect in many instances. Dental practitioners, on the other hand, have indicated a willingness to accept special needs patients. How is it possible that this contradiction has occurred? What can be done to bring the dental practitioner and the developmentally disabled patient together?

CASE FINDING

The first avenue is to become familiar with the types of educational, vocational and residential programs with which the developmentally disabled are affiliated. The early intervention programs service the 0-3 year age group. Services are primarily conducted in the home. However, many early intervention centers sponsor center-based programs as well. Participants attend these programs for approximately 2 1/2 hour sessions one to five days a week.

Chapter 766 of the Massachusetts General Laws mandates that special needs students receive a full range of educational and therapeutic services through the local school. These classes may be found in the local elementary and secondary schools. In other instances several local school systems join together to form an educational collaborative

to provide services to the special needs groups. In either case, the law requires that students receive these services from age 3-22 inclusive.

Upon graduation from their respective educational programs, special needs individuals will attend either a sheltered workshop, day activity, or day habilitation program depending upon the severity of the disability. Activities at these programs range from subcontract work from local industry to the teaching of basic activity of daily living skills.

There is also a residential component which provides services. For a multitude of reasons it is not possible for many clients to reside with their families, therefore, community group homes have been established. These may also be organized in different ways. Several individuals may live in one home with constant staff in attendance. Other individuals may live in supervised apartments in which staff may or may not be in constant attendance.

Contacting the agencies that provide these various types of programs to inform the directors that the dentist is interested in treating their developmentally disabled program participants would be the first step in locating potential patients. Of course, the dentist would have to advise the director of any special considerations that would preclude him from accepting all patients. For example, the office may not be wheelchair accessible or the dentist may not have hospital privileges to treat a patient who would need general anesthesia.

Many of these programs also periodically arrange meetings for the parents or responsible party to visit the facility. Speaking at a gathering of this type would provide families with the necessary information they would need when choosing a dentist. It would also provide an opportunity for families to meet the dentist prior to commencing any dental treatment. This could be very beneficial in allaying any fears or concerns families may have regarding dental treatment for their special person.

The dentist may also make known his presence and willingness to treat developmentally disabled persons by contacting the local advocacy organizations. There are a variety of advocacy organizations across the Commonwealth of Massachusetts which advocate for the service needs of their respective population groupings. These would include the Association for Retarded Citizens (ARC), United Cerebral Palsy (UCP), the Epilepsy Foundation, the Association for Mentally Ill Children (AMIC) as well as various advisory committees which deal with specific concerns of the many subgroups. Most of the advocacy organizations provide newsletters for their membership. Either writing an article on a specific dental manifestation of a particular developmental disability or simply requesting that your name and address be listed in a newsletter as being available to accept new developmentally disabled patients, would be another way to communicate a willingness to accept new special needs patients. Many of the advocacy groups also maintain a directory of local service providers including dentists. Having one's

name included in a directory of this type enables this information to be passed on to families who may inquire about dental referral information.

REFERRAL ISSUES

What are some of the barriers to care that have prevented the successful completion of dental treatment? There have been many reasons why the developmentally disabled have not received adequate dental treatment. These can be divided into two groupings, namely, barriers to care that originate from the dental profession and barriers to care that originate from the special patient population.

Perhaps one of the major obstacles to care has been the lack of formal training in the schools of dentistry and dental hygiene in the methodology for treating special patient populations. The Robert Wood Johnson Foundation several years ago awarded substantial grants to ten dental schools nationwide to develop curricula for inclusion into the dental school education. This has had a positive impact in resolving this barrier to care. Other training programs are also available to provide members of the dental profession with the skills necessary to treat certain special patient populations.

The major barrier to care originating from the special patient population has been a perceived scarcity of dentists who would accept the special needs family member. Surveys conducted by the National Foundation of Dentistry for the Handicapped and the Massachusetts Developmental Disabilities Council have indicated that there have been

an adequate number of dental practitioners willing to treat the developmentally disabled population. However, these practitioners may not be geographically accessible to the patient.

Another barrier of care has been the lack of public transportation that exists in many areas of the Commonwealth of Massachusetts. Even in those areas with public transportation there have been obstacles for those individuals who require wheelchair accommodations.

The third major obstacle concerns the financial implications of dental treatment. Although most adults are covered by Medicaid, many children are not. Futhermore, the medical program does not always adequately compensate dentists for their services. The Dental Treatment Fund administered by the Department of Public Health has addressed this issue. The Grottoes of North America, a subgroup of the Shriners, also has treatment monies available to assist families in paying for dental treatment.

The final obstacle, and perhaps the greatest, has been the apathy exhibited by clients and their families as to the need for obtaining dental care. Continual education must be provided to these individuals to create a desire for dental services. In conclusion, a concerted effort is necessary by all individuals to identify developmentally disabled individuals in need of dental services. This, in addition to a coordinated effort to resolve the obstacles to obtaining dental care, will enable the developmentally disabled person achieve better dental health.

OFFICE ACCESSIBILITY AND ENVIRONMENT

Robert Hammond, R.D.H., B.S.

Since multiply handicapped individuals face numerous obstacles in their daily living patterns, choosing a dentist with a physically accessible office is of primary importance. Historically, because of their limited mobility, handicapped individuals have not had access to certain buildings. But new federal and state regulations of the Architectural Barriers Board make it possible for the handicapped to enter newly constructed buildings plus old buildings that have been renovated to accommodate them.

After a dentist has been chosen and has agreed to treat a handicapped person, a preliminary visit by that person or someone who will be bringing the patient to the office should be made. If this is not possible, at the initial contact, inquiry should be made regarding access. Any problems of access should be explained to the dentist as he or she may have some useful suggestions. If the patient uses a wheelchair or has certain motor problems, steps and narrow doors present barriers to that individual.

Since regulations exist requiring that the primary public entrance(s) of a building be accessible to persons in wheelchairs, the approaches to such entrances are of primary importance. Entrances must be paved with a ramp of a non-slip surface, uninterrupted by stairs or steps. Adequate space must be provided, (sixty (60) inches from the door), on the interior and exterior, to allow free movement of the door without impeding a person in the wheelchair. If there are two doors present, a space of forty-eight (48) inches plus the width of the

swinging door should be available in that vestibule. The accessible entrance should also be on a level which permits access to building elevators where provided.

When there have been ramps constructed, either outside or inside the building, the handicapped person does have an easier time entering that building. These ramps must have specified widths and angulations. The width shall not be less than forty-eight (48) inches minimum clearance, mearsured at the railings. The slope of the ramp shall not exceed one (1) inch for every twelve (12) inches of angulation. Handrails shall be constructed on both sides of the ramp at two different heights, one of thirty-four (34) inches and a lower one at nineteen (19) inches, measured vertically from the surface of the ramp. These handrails must extend at least twelve (12) inches beyond the top and bottom of the ramp, but they should not so extend if it would cause a safety hazard. Ramps must have a surface that is non-slip, and they should not be carpeted except with certain specified low-pile, non-absorbant material. Each ramp should have level platforms for turning and resting and these platforms must be unobstructed by door swings, entrances and other projections. Such platforms must exist at intervals of thirty-two (32) feet along the ramp or wherever a ramp changes direction, plus at the top and bottom of the ramp. Circular ramps are not acceptable and must have the approval of the Architectural Barrier Board.

It is important to remember that any entrance to a facility not accessible by persons in wheelchairs should have a sign clearly indicating the accessible entrance.

If the patient is not confined to a wheelchair and can avail themselves to using stairs, please keep in mind that there are certain specific details to take into account. Any stair riser must slope no more than one and one-quarter (1 1/4) inch from the horizontal projection. Handrails must be provided on both sides of the stairs. Stair treads shall have a non-slip surface, and only a non-slip finish may be applied.

Any floor, be it inside the dental office, in a hallway, or in a vestibule, should have a common level throughout a single story except where a ramp connects different levels. Floors should have a surface that is non-slip and should be maintained with a non-slip material. If carpeting is used, a commercial or industrial type is recommended. The carpet should have a low pile and be non-absorbant. It should also be stretched tightly and securely anchored at all open edges. If padding is installed, it should not exceed one-quarter (1/4) of an inch in thickness, and it should be stretched and secured tightly to the floor. Tile flooring is not recommended.

Within the dental office, there are no specific rules or regulations governing physical space, but certain general recommendations are advised. A brief explanation concerning doorways must be made. It is important, as far as wheelchair access is concerned, that doorways have a minimum width of thirty-six (36) inches, thereby allowing a minimum of

thirty-four (34) inches for clearance, taking into account doors and hardware.

Once inside the office, the handicapped patient should be guided to the operatory if they are ambulatory or if they have received pre-medication. For obvious reasons, an unsteady patient is at risk as far as sharp corners on cabinets or counters is concerned.

The operatory itself should have adequate space for maneuvering a wheelchair or physically compromised patient. Any operatory should be at least eight (8) to ten (10) feet in width, with a minimum of three (3) feet on either side of the chair for wheelchair access to movable dental chair arms. If the patient is to be transferred to the dental chair, the chair should be swung to whatever side is easier for the transfer. Portable instrument cabinets and handpiece units provide additional space by being moved easily.

Initially it should be decided whether to treat the patient in the dental chair or in their wheelchair. If the patient is verbal, ask them what they would prefer. If nonverbal, the person accompanying the patient may provide valuable information. In either case, always check first, so that the transferrance can be done as smoothly and as easily as possible. There are accessory headrests available that can be easily adapted to the handles of a wheelchair.

Once the patient is being treated, it is extremely important to stabilize them without providing undue physical restraint. These measures should be explained completely beforehand so that the patient

does not become frightened or apprehensive. Some examples are blankets, pillows, foam pads, etc.

With complete sharing of information between the dentist and the handicapped patient, a smooth transition to thorough and complete dental treatment can be realized. Making a handicapped individual's dental experience easier and more comfortable, produces a better patient and increases the dentist-patient relationship to a level of mutual trust and understanding.

Source of information:

Rules and Regulations

Architectural Barriers Board

General Laws, Chapter 22, Section 13A

ROLE OF THE DENTAL HYGIENIST IN CARE OF DEVELOPMENTALLY DISABLED PATIENT

Deborah J. Hofer, R.D.H., B.S., Ed.M.

As a primary care provider, the dental hygienist should be fully cognizant of each patient's medical, physical and emotional condition. The disabled patient, may present with a variety of systemic and/or physical factors that need to be considered, and which may require modification of the treatment to be rendered, as well as the time frame for completion. This is not to say that treating a disabled patient will greatly disrupt office routine, necessitate specialized training or require special equipment.[5] The "modifications" needed to treat the community-based developmentally disabled patient are quite often no more than developing a thorough patient assessment and medical consultation protocol, allowing extra time to perform clinical procedures and adjusting home care recommendations and expectations to levels realistic for the individual's condition and capabilities.

Since the dental hygienist is usually the first member of the dental team with whom patients come in contact, the hygienist may be called upon to assume the administrative duties of initiating an office protocol for treating disabled patients. While treatment procedures vary from office to office, the basic goal remains the same: helping the patient reach and maintain optimum levels of oral health and function. To help the hygienist (or dentist) devise an office treatment protocol, some key factors are examined below.

PATIENT ASSESSMENT AND MEDICAL CONSULTATION

In order to prepare for the disabled patient for dental treatment, it is helpful to obtain as much information as is possible prior to the

first appointment. Ideally, the patient and his/her parent or direct care staff would come to the office for a preliminary interview. This allows the hygienist to observe the patient's general appearance, degree of mobility and attitude toward dental care. At the same time, the hygienist would be able to evaluate the patient's medical and dental history for any conditions that would require prophylactic antibiotic premedication, special operator precautions, sedation, consultation with the attending physician, or emergency (palliative) treatment. If a preliminary interview is not feasible, the medical and dental history forms should be mailed to the patient (or the person caring for the patient) if s/he is not competent or able to answer for him-/herself), with the request that the forms be returned by mail prior to the first appointment. Depending upon the patient's disability, a follow-up phone call could then be made concerning any further questions.

A complete medical history asks questions concerning:

-major illnesses/hospitalizations

-current treatment by a physician

-physician's name, address and telephone number

-familial medical history

-prescribed (and over the counter) medications

-allergies

-communicable diseases

-cardiovascular disorders

-blood disorders

-seizure disorders

-sensory disorders

-physical disabilities[3, 4]

In addition, specifics salient to the patient's disability or disabilities should be ascertained:

-when did the disability occur and/or when was it diagnosed?

-what methods of treatment have been employed?

-what, if any, therapy is the patient currently undergoing?

-has the patient ever been institutionalized?

-is there any sensory involvement? how does the patient communicate?

-what is the patient's level of mental and physical function?[3, 4]

A complete dental history asks questions concerning:

-previous dental treatment received and date of most recent visit

-anesthetic agents used

-analgesic agents used

-adverse reactions to any medications above

-previous radiographic surveys (date of most recent radiographs; availability for previous dentist)

-traumatic injuries to the face or teeth

-preventive services received (fluorides, sealants, nutritional counseling, plaque control) and frequency

-chief compaint, or reason for seeking dental treatment at this time[3,4]

The data obtained from the subjective and objective interviews should be sufficient to determine the need for: prophylactic antibiotic premedication, operator health precautions, environmental safety precautions, consultation for possible drug interactions, and/or possible emergency treatment. However, if any question remains concerning the patient's condition, medical treatment or contraindications for dental procedures, a consultation with the attending physician is indicated. While the necessary information may be obtained from the physican over the telephone, it is recommended from a legal standpoint that a written confirmation of the consultation be obtained.

Wheelchair to Dentalchair Transfers

Technically, patients who rely on a wheelchair for mobility have a well-practiced, preferred method for moving to other seating accomodations. The patient of guardian will usually be able to give detailed instructions on the transfer method that works best. In order that the hygienist have some prior knowledge of how wheelchair to dental chair transfers are accomplished, three general maneuvers are outlined below. In each case, a pathway at least 3 feet wide should be cleared into the operatory and next to the dental chair, which should be raised or lowered to the same height as the wheelchair. The wheelchair should be locked into into position parallel, and as close as possible, to the dental chair.

1. "Slide" - as the name implies, the patient simply slides from one chair to the other. Depending upon the patient's upper body strength, this maneuver is accomplished with or without a "transfer board" (small, polished board that bridges the gap between chairs and facilitates movement between the two)

2. "Stand and Pivot" - the patient is assisted to a standing position (by supporting the patient under the armpits; the patient's arms go around the assistant's neck), pivoted in position so that his/her back side is facing the dental chair, then gently assisted into a sitting position again.

3. "Lift" - patients who are unable to move or assist in moving themselves must be lifted up and over, into the dental chair. If the patient is small and light enough, this can be accomplished by one person alone holding the patient under the knees and supporting the back, with the patient's arm around the assistant's neck. For a bigger, heavier patient, two people will be needed to complete the transfer; one lifting the patient's upper body by supporting the trunk under the armpits from behind, while the

second person lifts the lower body by supporting the legs with both arms around the knees from the side.

Patient Stabilization

Some individuals with developmental disabilities require stabilization of their mouth and/or their body in order to receive dental treatment. For these patients, as well as patients who exhibit anti-social behavior, stabilization devices such as mouth props, velcro straps or a papoose board may be needed. When using stabilization devices such as these, it is important to reassure the patient that they are not being punished, but rather that the device is being used to help make treatment easier for them.

TREATMENT PLANNING

In order to formulate a treatment plan, clinical data from a thorough extraoral and intraoral examination dental charting, periodontal charting, orthodontic assessment and radiographic survey is needed. These data gathering procedures have traditionally been delegated to the dental hygienist. The dentist then interprets the clinical and radiographic findings to form a diagnosis and plan for the appropriate rehabilitative and/or preventive services.

The dental hygienist is also called upon for input when planning the preventive phase(s) of treatment. In order to accurately help the dentist plan the integration of preventive services (scaling, root planning, polishing, fluoride application, plaque control, nutritional

counseling, and/or placement of sealants) into the treatment plan, it is important that the hygienist also be aware of any limitations a patient's handicap may place on the ability to receive treatment.

While most routine instrumentation and education procedures will remain the same as for "normal" patients, many disabling conditions will require that appointments be scheduled at specific times of the day. Morning appointments may be indicated for patients who tire easily (as do patients with many neuromuscular disorders), or who suffer from certain medical conditions such as the insulin dependent diabetic (who would optimally be seen 1 1/2 hours after breakfast and/or taking the prescribed medication.)[1]

The length of each dental appointment is another factor that may have an increased significance for the disabled patient. Decreased physical stamina and/or anxiety often experienced by patients prior to treatment may dictate the need to schedule a series of short appointments to complete treatment that would ordinarily be done in fewer, longer appointments.

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